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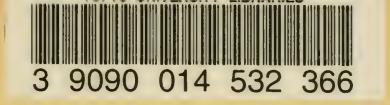
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A MANUAL

OF THE

THEORY AND PRACTICE

OF

EQUINE MEDICINE.

BY

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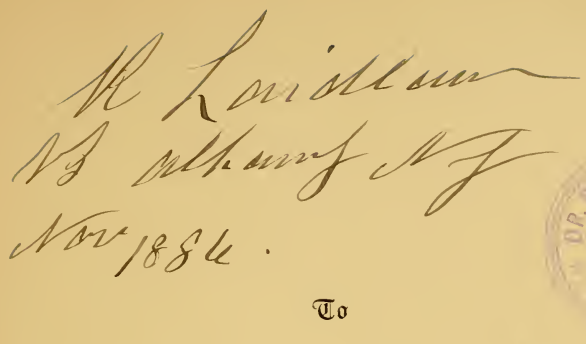

To

HENRY CHAPLIN, Esq., M.P.,

THIS LITTLE MANUAL IS

Respectfully Dedicated,

AS A SLIGHT TOKEN OF ESTEEM TO ONE WHO,

AS A STATESMAN,

HAS DONE SO MUCH TO PROMOTE THE

INTERESTS OF AGRICULTURE;

AND IN GRATEFUL RECOGNITION OF PAST KINDNESSES,

BY

THE AUTHORS.

R. Laidlaw 15
Oakhurst N.J.
November 7th
1884

PREFACE.

In preparing the first of these Manuals of Veterinary Science, it has been our object to bring the chief facts of Equine Medicine and Pathology before the reader in as concise and clear a manner as possible.

It is our hope, regarding the many and great difficulties which are necessarily encountered in a work of this kind, that allowance will be made for errors of omission and commission which may be detected.

We have adopted a somewhat different classification of disease from that in vogue, and trust that its greater simplicity will entail less labour on the part of the student, without rendering it any the less scientific.

Throughout we have endeavoured to lay stress on those points which are of very great interest and importance from a comparative pathological point of view.

Into the histological structure of the malignant growths of the horse we have, purposely, not fully entered, it being our purpose to publish, as soon as opportunity arises, a monograph on the comparative pathology and etiology of the malignant growths in man and animals.

In the treatment of disease we have followed more especially the methods adopted by the late Mr. D. Gresswell, F.R.C.V.S., and those which practical experience leads us to consider of the greatest value.

We have appended chapters on Entozoa and Toxicology, as we consider both these subjects of great importance to the veterinary practitioner.

VETERINARY INSTITUTION,
LOUTH, LINCOLNSHIRE.
November, 1884.

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DR. R. LAIDLAW, V.S.
ALBANY, N. Y.

A MANUAL OF THE THEORY AND PRACTICE OF EQUINE MEDICINE.

INTRODUCTORY.

THE Ungulata, or Hoofed Quadrupeds, one of the largest natural orders of the Mammalia, are classified into two sub-orders, according as the animals have an odd or an even number of toes on the hind-foot. Those which have an odd number on the hind-foot are termed Perissodactyla (περισσος, odd, and δακτυλος, toe), and those which have an even number are termed Artiodactyla (αρτιος, even, and δακτυλος, toe).

To the latter group belong such animals as the Pig, Hippopotamus, Sheep, Ox, Camel, Giraffe, and Deer. The Tapir, Horse, and Rhinoceros make up the former group.

The Artiodactyla are divided into two classes: the Ruminantia, and the Non-Ruminantia.

The Ruminantia include the Sheep, Ox, Camel, Giraffe, and Deer; and the Non-Ruminantia include the Pig and Hippopotamus.

All the Ungulata, excepting the Pig and Peccary, which are omnivorous, feed upon vegetables. Their placentation is non-deciduate, and in this they agree with the order of the Cetacea; and they are always adapted for swift progression.

The sub-order Perissodactyla comprises three living families: the Tapiridæ, or Tapirs; the Equidæ, or Horses; and the Rhinocerotidæ, or Rhinoceroses. There are also several extinct families: the Macraucheniadæ (μακρος, long, and αυχην, neck); the Palæotheres (παλαιος, old, and θηριον, beast); and the Lophodontia (λοφος, a crest, and ὀδους, a tooth).

The Perissodactyles have no fewer than twenty-two dorsi-lumbar vertebræ. They have a third trochanter on the femur, and the third digit of each foot is symmetrical. Horns, when present on the head, are only skin deep, are always placed on the middle line of the skull, as in the Rhinoceros, and have no bony core.

The number of toes in the Perissodactyles is reduced to a minimum.

The hind-foot of the Tapir has three well-developed toes, and these are homologous with the second, third, and fourth digits of man.

In the Rhinoceros all the feet are provided with three toes formed of the same three digits.

In the Horse there are further reductions; for that digit which corresponds with the third or middle digit of man is the only one which remains intact. There are, however, two rudimentary digits in addition, and these are called the splint-bones. They are homologous with the second and fourth digits of man.

The Perissodactyles are first represented in geological strata of the Old World by the Lophodontia. These extinct forms, which are found in the Eocene strata of France and England, were closely allied to the Artiodactyles. As in the latter, their pre-molar teeth were simpler and longer than the molar teeth. The feet of the Lophodons, however, were of the Perissodactyle type.

Of all existing Ungulate animals, the Lophodon comes

nearest to the Tapirs, which are the most lowly differentiated of existing Perissodactyles.

The Lophodon is hence regarded as the ancestral form of the modern Tapiridæ.

The Acerotherium is the earliest representative of the Rhinoceros, and is met with in the Miocene strata of the Old World.

The Palæotheres first occur in the Eocene strata of Europe and North America. They varied in size from that of a Roedeer to that of a Tapir, and their molar teeth resembled, in the pattern of the grinding surface, those of the Rhinoceros.

They are allied on the one hand with the Horse and Rhinoceros, and through the Macraucheniadæ on the other with the Camels and Tapirs.

Palæotherium Magnum was of the same size as a Horse, and Palæotherium Medium of the same size as a Pig.

In the Eocene strata of the Utah Territory of North America is found the Eohippus. This extinct animal is the earliest ancestral form of the Horse. It was of about the same size as a Fox, and its feet have three toes behind and three in front.

In the early Miocene and late Eocene the Anchitherium appears. It was of the size of a sheep.

In the early Pleiocene and late Miocene the Hipparion, Protohippus and Pleiohippus appear. These extinct forms were about the size of an Ass.

The Hipparion was a small graceful animal, having three well developed toes, each bearing a hoof. The middle toe was strong and large, while the lateral toes were so small as not to reach beyond the fetlock.

It is noteworthy that cases are recorded where Horses have been born with a tridactyle foot in every respect similar to that of Hipparion.

The Equidæ comprise several living species. They are divided into the true Horses, which have horny patches or callosities on the inner side of both pairs of limbs, above the wrist on the fore, and on the inner side of the metatarsus on the hind limbs; and the Asses, which possess such callosities on the fore limbs only. With the Asses are classed the Zebras and Quaggas. The true Horses are represented by one well-established species, Equus Caballus, from which all other races or varieties are descended, through the gradual processes of natural and artificial selection.

These races vary as much as any two closely allied species of wild animals.

According to Mr. Darwin, no aboriginal or truly wild Horse is positively known to exist, for the wild Horse of the East may probably be descended from those which have escaped from domestication. Probably the wild animals have been exterminated by the hand of man in those countries which they formerly inhabited, and in which they have left their remains to attest their former presence.

The wild horses of Tartary present us with the nearest examples of the stock from which the domestic horse is probably descended. These wild animals have a stripe along the back, and are of a greyish drab colour.

The first horses known in Europe were introduced in the period known as the Neolithic Age. They were in all probability used for food in this age, and not employed for driving or riding.

In the succeeding or Bronze Age they were probably introduced from the Steppes of Central Asia, and were used for riding and driving, as is evidenced by the discovery of bronze bits.

In the Scriptural records the Horse is first mentioned

during the Egyptian famine, and it was probably very similar to the Arabian Horse of the present day.

From the earliest ages known to historians, horses were used for war in chariots. To the early Greeks the art of riding was unknown down to the year B.C. 500. At the time of the Grecian supremacy, however, horses were used for the chariots, and about the year 450 B.C. we read of riding and cavalry in Greece Proper.

If we turn to Roman history, we learn that in the early Regal times, and in the first centuries of the Republic, cavalry was the most important weapon of the military armament.

The ancient Britons used horses for their chariots in their wars against the Romans, and representations of horses occur on some of the early British coins.

Athelstan made an enactment to prevent the exportation of horses, and imported *running* horses from Germany, and also horses from Spain, in order to improve the native breed.

King John paid much attention to breeding horses for agricultural purposes, and, according to Youatt, we are indebted to this monarch for our Draft Horses.

Edward III. improved the breed by mixture with lighter horses of Spanish breed. From this time, owing to the great care taken in breeding, races were regularly established in various parts, and enactments were made by various sovereigns from time to time, in order to secure excellence.

In the Protectorate of Cromwell, horses were introduced from Morocco and the South East; and by this means elegance of form and swiftness were added to the stoutness which had hitherto characterized the English horse.

The original colour of the horse was a dun-brown.

Thus artificial selection by man, in addition to the action

of natural selection, has been of very great importance in the establishment of the various breeds of horses.

It is indeed remarkable how the forms of horses have been modified by the various conditions of life. As an example, it is well-known how horses living in mountainous districts, or small islands, become reduced in size from deficient supply and want of variety of food.

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CHAPTER I.

ON THE CAUSES, SYMPTOMS, AND CLASSIFICATION OF DISEASE.

ETIOLOGY.

By etiology (αἴτιος, cause, and λογος, law) we mean the science of the causation of disease.

Etiology is of great importance, it aids our diagnosis, our prognosis, our treatment, and our prophylactic measures. The causes of disease may be divided into intrinsic and extrinsic causes.

INTRINSIC CAUSES of disease are :

1. Heredity.—Offspring may inherit from their parents an actual disease, or they may, as far oftener occurs, inherit a special tendency to various forms of disease.

2. Age.—Certain diseases are especially characteristic of old age, while others are more liable to attack young animals.

3. Sex.—Besides the diseases arising from the peculiarities of the generative organs of the two sexes, it has been observed that sex is of influence in predisposing to certain forms of disease. We may give, as an instance, roaring, which is more common in horses and geldings than in mares.

4. Variety.—Some breeds of animals are more liable to certain diseases than others. As an example, it has been

found that navicular disease is rare in cart-horses, though it is a common affection among the more highly bred animals. Again, roaring is very often associated with certain shapes of neck.

5. **Colour.**—Melanoid sarcoma is frequent in grey or white horses.

6. **Temperament** predisposes to certain diseases. The sanguine temperament is especially shown in the highly bred horses, whereas in lower bred animals the lymphatic temperament is predominant. The sanguine temperament predisposes to diseases of an inflammatory type.

7. **Diathesis.**—By this term we mean a special liability to certain diseases; for example, a rheumatic or tubercular susceptibility.

8. **Effects of Previous Disease.**—Some diseases when once acquired tend to recur; others, again, are *protective against themselves.*

EXTRINSIC CAUSES of disease are:

1. **Dietetic.**—Food and water are frequently sources of disease, when excessive or deficient in amount, or when the quality is unsuitable.

2. **Atmospheric or Telluric Influences.**—These are of much importance in the production of disease, as are also the locality and geological formation.

3. **Imperfect Sanitation.**—Defective drainage and imperfect ventilation are fertile sources of disease; *indeed, there is no more potent cause of disease among horses than insufficient or vitiated supply of air.*

4. **Work.**—When too severe, work is a cause of disease, as also it is when insufficient.

5. **Temperature.**—Heat and cold are of much influence in the causation of disease.

6. **Mechanical Causes.**—Injuries of various kinds.

7. **Poisons.**—Mineral poisons, as arsenic, antimony, and

lead; and vegetable poisons, as the yew tree, hellebore, and the rhododendron.

8. Parasites—Animal and Vegetable.—Animal parasites will be considered in detail in a later chapter.

Vegetable parasites are most potent sources of disease. When treating of Bacteria we shall see that these vegetable fungi have been demonstrated in the blood and tissues of animals suffering from many diseases. As an example, anthrax or charbon will for the present suffice.

In addition, parasitic fungi are frequently developed on the external parts of the body, producing definite disorders, but not invading the system at large.

The so-called Tineæ are examples of such diseases. They are produced by the growth of parasitic fungi, such as the Trichophyton tonsurans and Achorion Schönleinii.

SEMEIOLOGY.

By semeiology (σημειον, sign, and λογος, law) we mean the science of the signs or symptoms manifested by the diseased animal.

Symptoms are local or general, according as they are restricted to a special part or affect the system as a whole.

They are called primary when they arise directly as a result of the disease; sympathetic or secondary when arising from those caused by the primary disease or from a secondary disorder.

They are called premonitory when they precede the full development of the disease. And symptoms are still further divided into diagnostic, prognostic, and therapeutic, according as they are respectively applicable to the distinction, the determination of the result, and the suggestion of the treatment, of the disease.

NOSOLOGY.

By nosology (νοσος, disease, and λογος, law) we understand the definition, the naming, and the classification of the diseases.

It is at present impossible to frame a perfectly natural classification of the diseases of man and animals, as our knowledge is inadequate for the purpose; but with the advance of medical science our system of classification will doubtless become gradually more and more perfect.

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CHAPTER II.

GENERAL PATHOLOGY.

CONGESTION, OR HYPERÆMIA.

CONGESTION, or hyperæmia, is excess of blood in the dilated vessels of any part. It is active or arterial, and mechanical or venous.

Active Hyperæmia is excess of blood in the arteries of any part, generally accompanied by acceleration of the blood-flow. It is caused by diminished arterial resistance, which may be due to several causes, viz. :

1. Weakening or paralysing of the coats of the vessels.

2. Inhibition of the tonic action of the sympathetic on the walls of the vessels, which may occur either directly or reflexly.

As an example of the direct process may be given the congestion occurring after section of the vaso-motor nerves, in any part of their course from their origin in medulla oblongata, to the spinal or sympathetic plexuses.

Some drugs also, as nitrite of amyl, tobacco, and alcohol, probably directly paralyse the vaso-tonic nerves.

The reflex process is due to stimulation of afferent nerves. It is seen when food excites the flow of saliva or gastric juice.

3. Excitation of the vaso-dilator nerves, as, for example, the chorda tympani.

Mechanical, or Venous Hyperæmia.—In venous hyperæmia the blood-flow is retarded, and the excess of blood is in the veins and capillaries.

Venous hyperæmia may be caused by—

1. Diminution of the force with which the blood should be driven through the veins.

2. Direct impediment to the return of blood by the veins.

We may consider these causes separately.

(1.) Diminution of the force of the circulation may be due to lessened cardiac power; for example, in the acute febrile disorders and in those degenerations of the cardiac structures which lead to dilation of its cavities.

In the arteries also the blood-current may be obstructed from morbid changes in their walls.

The current may also be obstructed in the capillaries, chiefly from the pressure of inflammatory effusions, dropsy, and other causes.

Finally, the flow may be impeded in the veins, owing to absence of muscular contraction of the walls, or to dilation of the vessels.

(2.) Direct impediment to the return of blood by the veins may be due to various causes.

As examples may be given the obstruction to the portal circulation which occurs in cirrhosis of the liver; the congestion of the lungs which occurs in mitral constriction and regurgitation; and that of the lower extremities which occurs from the pressure of the gravid uterus on the iliac veins.

The Results of Venous Congestion.—Venous congestion results in accumulation of blood, and diminished velocity of its flow in the dilated capillaries and veins. The most important changes which may follow are: transudation of serum, diapedesis of red blood-cells, hæmorrhage, fibroid induration, and frequently thrombosis. Gangrene, *i.e.*, death of the part, may result when the obstruction is very general and complete.

INFLAMMATION

May be defined as 'the succession of changes which takes place in a living tissue as the result of some kind of injury, provided that the injury be insufficient immediately to destroy its vitality' (Sanderson).

The phenomena of inflammation in vascular structures comprise—

1. Changes in the blood-vessels and circulation.
2. Exudation of fluid and blood-cells from the vessels.
3. Changes in the tissues affected.

(1.) **Changes in the Blood-vessels and Circulation.**—The first effect of inflammation is dilatation of the arteries. This dilatation steadily increases for about twelve hours, affecting chiefly the arteries, but also the veins. The capillaries are but slightly affected. The blood-flow becomes accelerated at first, and then in about an hour it becomes considerably retarded, though the vessels still remain dilated. Pulsation in the small arteries becomes manifest, and slowness of the stream allows of the distinction of individual corpuscles in the capillaries and smallest veins.

The acceleration of the blood-flow is not a constant occurrence, and often subsides without the appearance of the other phenomena of inflammation. The dilation, with diminished velocity, however, *is* constant, and is the essential vascular change in inflammation. As the blood-flow becomes retarded, white blood-cells gradually accumulate in the smaller veins, adhering to their walls, and some also stick in the capillaries.

Thus the lumina of the veins become narrowed by layers of white cells, among which there are no red ones; the circulation becomes slower and slower, and the red blood-cells, with some white ones, accumulate in the capillaries.

Eventually the blood-stream in the capillaries ceases,

and the accumulated mass of cells moves to and fro with the pulse. This is the *stage of oscillation*, and it is followed by that of *stasis*, in which no movement of any kind takes place.

Finally, thrombosis may occur, when the capillary walls have lost all vitality.

(2.) **Escape of Fluid and of Blood-cells from the Vessels.** —Some of the leucocytes adhering to the sides of the vessels gradually sink into the walls of the small veins, and (to a less extent) of the capillaries, and pass through them. Red blood-cells also pass through, but to a far less extent. In severe inflammation, however, where the stagnation in the capillaries is extensive, red cells may pass through almost alone, and in large numbers, thus giving the exudation an hæmorrhagic character. The red cells pass out chiefly from the capillaries; the intensity of the inflammation and the vascularity of the tissues determining the proportion of them in the exudation.

The red and white cells at first remain near the vessels from which they have escaped, but are soon washed away by the exuding fluid, and the white cells move, in addition, by their own peculiar power of locomotion.

It is almost certain that all new cells formed in inflamed tissues, as a direct result of the injury which caused the process, are *escaped blood-cells.*

In the less acute forms of inflammation, cells are also formed by regenerative processes going on in the cells of the tissues; but these are not of inflammatory origin. The composition of inflammatory exudation is not constant. In the most acute inflammations it contains very many red cells; but in the less severe forms, white cells are in excess of the red.

The more severe the process, the more does the exudation resemble plasma in composition and character; while in the less severe forms it becomes more like the fluid exuded in mechanical hyperæmia.

(3.) **Changes in the Inflamed Tissues.**—Inflammation leads to depression of vitality, degeneration and death of the tissues involved. No increased vitality and no multiplication of tissue elements, form part in the process.

The essential lesion of inflammation is some change in the vessel-walls themselves. That these are affected is shown by the facts that all the early phenomena of inflammation are vascular, that injury of the vessels alone causes inflammation, and that injury of the tissues alone (Senftleben) does not cause them.

Furthermore, Ryneck has shown that stasis may be produced in the vessels of the web of the foot of the frog, in which milk or defibrinated blood is circulating in place of normal blood; and that no such stasis can be produced in vessels whose vitality has been destroyed by the injection of metallic poisons.

In all spontaneous inflammations the cause is first probably carried to the part by the blood, and acts firstly upon the vessels, and afterwards upon the tissues. There is no detectable structural alteration of the vessels, and so Cohnheim speaks of the change as molecular, and regards it as possibly chemical in nature.

Clinical Signs of Inflammation, and their Causes.—The clinical signs of inflammation are redness, heat, swelling, pain, and impaired function.

Redness and heat depend upon the amount of blood passing through the part in a given time. *Heat is not produced in excess* in the inflamed tissues.

Swelling arises from exudation of fluid and cells. It may be entirely due to fluid, as in hydrocele, or to cells mainly, as in orchitis, where the fluid has been absorbed.

Pain is due to pressure of the effusion on the nerve-endings, and perhaps also to chemical irritation of them. The

more sensitive and rigid the tissue, and the more rapid the effusion, the greater is the pain.

Impaired function is due to the injury of the tissues involved.

VARIETIES OF INFLAMMATION.

The several varieties of inflammation are to be regarded simply as steps in the process, due to differences in

1. The resisting power of the tissue;
2. The intensity of the cause; and,
3. The duration of its action.

Serous Inflammation.—The best examples are chronic effusions into serous cavities, as the pleura, tunica vaginalis, or the joints. In impoverished states of the blood, especially when the albumen is diminished, inflammatory exudations are liable to be serous, even though the process be of considerable intensity. In serous inflammation the exudation contains excess of albumen, and but few white cells; hence it does not coagulate, though a few flakes of fibrine may be formed.

If the process be more severe, the exudation contains more albumen, fibrinogen, and white cells; and hence fibrine forms in increasing quantity.

Networks of fibrine are of frequent occurrence in the meshes of inflammatory connective-tissue. These inflammations are termed sero-fibrinous.

Fibrinous Inflammation.—In this variety the exudation is still more richly albuminous, and contains more leucocytes. It has a much greater tendency to coagulate, and 'lymph' forms on the inflamed surface, or in the substance of the inflamed tissue.

The best examples of this kind of inflammation are those of serous membranes, the pleura for example. Here fibrine containing leucocytes is exuded, and is termed 'lymph.'

'Lymph' may form on both the visceral and parietal

pleura, and the two opposed patches of inflammation may blend. This is the first stage in the formation of adhesions, *i.e.*, bands of connective-tissue between the two surfaces.

Lymph, similarly formed and composed of fibrine and leucocytes, is the temporary uniting medium in healing by the first intention.

This form of inflammation may end in absorption.

Productive Inflammation.—Often the inflammatory process ends in the formation of new tissue, and is then termed 'productive.' In these cases the fibrine disappears from the exudation, and white cells crowd into it, converting it into a tissue of closely packed white cells, in a scanty homogeneous matrix, which is supplied by vascular loops springing from the capillaries of the inflamed tissue, penetrating it in all directions. This tissue is called *granulation-tissue.* The granulations, when plentifully supplied with newly-formed vessels, gradually become converted into connective-tissue, or *scar-tissue.*

This tissue is at first very vascular. Gradually the new tissue contracts, many of the vessels disappearing in consequence, and eventually the scar-tissue becomes less vascular than the surrounding parts.

Granulation-tissue may, however, 'break down into pus,' instead of forming scar-tissue. This is due to the insufficient development of vessels, diminution in their lumina, continuance of excessive irritation, and to the too dense packing of cells.

The term **interstitial** is applied to inflammations of solid organs where the connective-tissue is chiefly involved.

The term **parenchymatous** is applied to inflammations where the epithelial cells of the organs show the most marked changes.

Suppurative Inflammation.—Is a very common form.

In this variety the exudation contains the same elements as does the fibrinous exudation.

No coagulation occurs in this kind, no 'lymph' forms and vascularizes. Any 'lymph' which may be formed at an early stage is destroyed.

The irritation is more intense than that required to produce a fibrinous exudation, and its action is more prolonged.

Suppuration may occur in the tissues in a circumscribed form or abscess or in a diffuse manner, or its seat may be a free surface, as a mucous membrane or the skin. In the latter cases, when the epithelium is destroyed, with more or less of the subjacent tissue, the process is termed *Ulceration*, but when the deeper layers of epithelium remain it is termed a purulent catarrh.

Formation of an Acute Abscess.—Fluid and corpuscles escape into the irritated tissue. The cells accumulate especially round the small veins, which thus form commencing foci of infiltration. The infiltrations become denser and denser, and spread by migration and transportation of cells until the foci blend. In consequence of the extent of injury and succeeding nutritive disturbance, the tissue elements die and disappear before the leucocytes.

In the central parts of the diseased area no vessels are to be seen, and no new ones are formed.

From mutual pressure and absence of nutrition, the central cells die and degenerate together with the intercellular substance. Thus a cavity containing dead leucocytes (pus-cells) is formed, and it is bordered by still living infiltrated tissue.

The process gradually spreads by thrombosis of the vessels and consequent disintegration of the cells they supply. An acute abscess almost always extends until it bursts or is opened.

If the cavity be kept at rest and drained, the inflammatory cell-infiltration vascularizes, and the walls thus become lined by granular tissue.

This grows and stretches across the cavity, and then develops into scar-tissue. In this manner the abscess is healed.

Diffuse Suppuration is a similar process going on in a wider area.

Suppuration is characterized by the formation of *pus*, which from an otherwise healthy animal is thick, creamy, opaque, yellowish-white, and slightly viscid, and has a faint odour and alkaline reaction. The specific gravity varies from 1030—1033.

It contains about 10—15 per cent. of solid material, of which two-thirds are albumen, the rest being fatty matter and salts, such as are found in the blood. On standing, it separates into a clear supernatant fluid, called 'liquor puris,' and a dense yellow layer of pus-cells.

Pus-cells are spheroidal, semi-transparent, more or less granular motionless cells, usually containing a bi- or a tripartite nucleus. They are about $\frac{1}{2800}$ of an inch in diameter.

The more recently escaped cells perform amœboid movements, and have the appearance of leucocytes.

Ulceration.—Is due to the same molecular destruction of the tissue on a free surface as was described above. Commonly, shreds of disintegrated tissue adhere to the floor of a spreading ulcer. In more intense inflammation the shreds may become much larger, and are thus transformed into sloughs.

Ulceration passes insensibly into gangrene, when death becomes too rapid to permit of molecular disintegration of the tissues involved.

When the inflammation ceases, the round-celled infiltration on the floor becomes vascularized into granular tissue.

These granulations are bright-red rounded elevations, about the size of pins' heads, and consist of a group of cells round a capillary loop. They have no lymphatics or nerves, are not tender, and do not bleed readily.

Hæmorrhagic Inflammation.—This form is characterized by an exudation in which red blood-cells are in great excess. The greater the vascularity of the tissue, and the more severe the injury, the more likely is an exudation to be hæmorrhagic. In acute pneumonia, the exudation generally contains many red cells. This form of inflammation too often terminates in gangrene.

TERMINATION OF INFLAMMATION.

Inflammation may terminate in resolution, in necrosis, or in new growth.

1. Resolution.—This is the commonest and most favourable termination of inflammation. It consists in the cessation of the process, and the restoration of the parts to the normal condition.

The corpuscles of the stagnant blood gradually move away until the current is re-established in the inflamed area. Migration of cells and exudation of fluid ceases, and the circulation becomes normal. The exuded fluid and cells are removed firstly by the lymphatics, and afterwards by the veins also. The remaining blood-cells or fibrin undergo fatty degeneration and are removed.

2. Necrosis.—Inflammation may terminate in death of the implicated part. The more severe the injury, and the feebler the resistance of the tissues, the more likely is necrosis to occur. We apply the term 'necrosis' not to the molecular disturbance occurring in suppuration and ulceration, but to obvious death of tissue, and generally *en masse*. Some affections, *e.g.*, malignant pustule, always

terminate in necrosis or gangrene. Such inflammations are termed gangrenous or necrotic.

Diphtheritic is a special variety of necrotic inflammation. It affects the surfaces of mucous membranes and wounds.

In this form the mucous membrane is covered by a false membrane, composed of a network of fibrin, containing here and there leucocytes in the meshes.

The epithelium is quite destroyed, with more or less of the subjacent tissue. This form of inflammation characterizes the disease called 'diphtheria,' but a similar pathological change may occur on any mucous membrane, *e.g.*, that of the intestine (dysentery).

3. New Growths.—Inflammations ending in new growths are the so-called productive inflammations already considered.

ETIOLOGY OF INFLAMMATION.

Etiologically, inflammations are divided into simple or traumatic, and cryptogenetic.

1. Simple or Traumatic Inflammations.—Are due to some injurious agency, such as mechanical violence, action of chemicals, excess of heat or cold, or to prolonged local anæmia. Inflammation from these causes has no tendency to spread beyond the part originally injured, or to pass on to more advanced stages, after the causes have ceased to act. Under this heading come inflammations due to wet or cold, 'rheumatic' and 'reflex' influences.

Excessive functional activity is said by some to be a direct cause of inflammation.

Nervous influence is also regarded as a direct cause.

2. Cryptogenetic Inflammations.—In very many of the inflammations there has been no obvious chemical or mechanical injury. Many of these have been shown to be due to the action of various vegetable fungi, and evidence

has been given to show that the others are probably due to the same cause. These inflammations are termed cryptogenetic. The fungi may act as chemical or mechanical irritants, thus acting in the same way as the causes of simple inflammation. When the products of the life-action of these low vegetable organisms are irritant, inflammation results; the intensity of the process varying with the quantity and intensity of the irritant. If the irritation is intense, suppuration results; if it is less severe, the early stages of productive inflammation occur, as is supposed to be the case in farcy and tubercle. The characteristic lesion of these and some other diseases is an inflammatory nodulate, raised round a spot at which the fungi have probably lodged, and whence they may spread and infect other parts. These lesions are spoken of as the 'Infective Granulomata,' the name signifying infective tumour-like formations of granulation-tissue (Ziegler).

Some fungi cannot live in healthy tissues; these are called 'non-pathogenic.' Of the others, which are called 'pathogenic,' some spread only by continuity of the tissues; others find a suitable nidus in lymph; whilst others enter the blood-stream, and are carried by it through the body (*e.g.*, Bacillus Anthracis). It is in this way that metastatic inflammations are to be explained; for example, the secondary abscesses of pyæmia, which constantly contain microbes, and the albuminuria, which so often complicates certain specific diseases.

The cryptogenetic inflammations are divided into the septic and the infective:

1. **Septic.**—Of non-pathogenic organisms, those which cause ordinary putrefaction are of much importance in inflammation.

The term **Septic** should be restricted to inflammations of this kind.

Often the discharges or sloughs from a simple wound, which has not healed by first intention and is not treated antiseptically, become putrid, and Bacterium Termo, and other organisms, especially micrococci, are found in them.

The products of the life of these fungi are the cause of the inflammation, and these organisms may continue to grow and excite fresh decomposition. If, however, the discharges are drawn off as fast as they are formed, the non-pathogenic organisms lose their pabulum and putrefaction ceases. Practically, if a wound be guarded from putrefaction, it is protected from infective organisms.

2. Infective.—Pathogenic organisms cause inflammation by penetrating into living tissues and causing irritant decomposition of the fluids. They are called infective because their poison multiplies in the tissues and spreads to near parts (locally infective), or distant parts (generally infective), in which they set up similar processes. Simple and septic inflammations are non-infective. The special peculiarities in the poisons cause special peculiarities in the inflammations, which are called 'specific' because they arise from specific causes.

THROMBOSIS.

By thrombosis we mean any coagulation of the blood in the heart, or vessels, occurring during life. The coagulum itself is called a thrombus, in opposition to a clot, the result of post-mortem coagulation. Thrombosis most commonly occurs in the veins. A thrombus may be distinguished from a clot formed after death by its laminated structure; its greater amount of fibrin, which makes it more firm and light than post-mortem coagula; its granular appearance on breaking; the greater relative proportion of white blood cells in it; and by its firmer adhesion to the part with which it is in contact. In all cases where coagulation has been very

rapid, the thrombus first appears on the lining membrane of the vessels, and slowly grows from without inwards by deposit of fresh fibrin, and thus finally blocks up the lumina of the vessels entirely.

It now extends in a direction towards the heart until it meets a current of blood strong enough to arrest its further progress.

CAUSES OF THROMBOSIS.—For the coagulation of blood three factors are necessary, viz., fibrinogen, fibrinoplastin, and the fibrin ferment. Fibrinogen exists as such in the liquor sanguinis. The ferment and most of the fibrinoplastin are contained in the white cells, the destruction of some of which is therefore necessary for coagulation. The causes of thrombosis are:

1. **Retarded Blood flow.**

2. **Modification of the Vascular Walls**; which consists in abnormality, or removal of the endothelium.

The causes which produce such modifications are:

A. **Injuries**: mechanical, chemical, or physical.

B. **Foreign Bodies**: the presence in the vascular system of substances not covered by endothelium, *e.g.*, needles, parasites, and new growths which have penetrated into the veins.

C. **Such changes in the Blood**, or in the supply of blood to a part, as cause disease of the vessel-walls, by imperfect or improper nutrition.

D. **Certain Conditions of the Blood.** The tendency to coagulation of the blood is said to be in some measure increased in the later months of pregnancy and after profuse hæmorrhages. The increased tendency of the blood to coagulate is probably never more than a predisposing cause of thrombosis.

CHANGES IN THROMBI.—These are decolorization (when red), resolution, organization, calcification, softening (simple and infective), and putrefaction.

Organization is commonest in arteries, while softening is chiefly met with in the veins.

RESULTS OF THROMBOSIS.—The coats of the vessels are altered, and unless collateral circulation be established, thrombosis of an artery will lead to death of the parts supplied. Thrombosis of an important vein will cause painful swelling; if continued long, nutritive changes in the parts it drains, and mechanical hyperæmia, will follow.

A fragment of the thrombus may be washed away by the blood current, thus leading to embolism.

EMBOLISM.

Embolism is the impaction of solid substances circulating in the blood-vessels which are too small to allow them to pass. These solid substances, which vary much in their nature, are termed emboli. The most frequent causes of embolism are detached fragments of thrombi in the veins, vegetations, and morbid *débris* from the heart, portions of new growths and parasites.

An embolus which arises in the systemic veins will probably be arrested in one of the small branches of the pulmonary artery; while those arising from the pulmonary veins, or heart, will be lodged in the systemic arteries, and those from the radicles of the portal vein in the liver.

Thus the seat of embolism is either in the arteries or in the hepatic branches of the portal vein.

The arrest of the embolus, and the consequent obstruction, are followed by the formation of secondary thrombi behind and in front of it. The secondary thrombi extend as far as the entrance of the first large collateral vessels.

Emboli may in rare cases be absorbed. When derived from thrombi they may organize or soften.

RESULTS OF EMBOLISM.—An embolus cuts off the supply of blood from the area to which the vessel is dis-

tributed, and deprives the part of its function, for the time, at any rate.

When the embolus is small, the collateral circulation is soon established, and the part may soon regain its normal condition. In other cases collateral congestion occurs, and a backward current takes place in the veins, leading to hæmorrhage.

The thrombosed tissue, infiltrated with extravasated blood, is called an hæmorrhagic infarct.

If the infarct be infective, rapid softening and disintegration takes place and an embolic or metastatic abscess is formed.

If the infarct be non-infective and small, absorption or organization takes place. If the infarct be non-infective and large, it often softens and then becomes encapsuled, and finally dries up, leaving a fibroid cicatrix.

CAPILLARY EMBOLI.—These consist of fat, masses of organisms, white blood-cells in small masses, pigment-granules, or air.

In fractures, contusions of subcutaneous tissue, acute osteo-myelitis, and other marked conditions in which fat-cells are broken up and the fat set free, the particles are absorbed and carried away by the lymphatics and veins.

These emboli reach the right side of the heart, whence they are carried to the pulmonary arterioles and capillaries. These little plugs may be gradually swept on to the left side of the heart, and are thence distributed by the circulation to other organs, where they may collect in large numbers.

HÆMORRHAGE.

Hæmorrhage signifies an escape of blood from the general current of the circulation.

It may arise from rupture of vessels due to injury or to

disease of the walls, or to overfulness or increased tension, as in congestion.

Hæmorrhage may also be due to alterations in the quality of the blood, as in purpura hæmorrhagica.

The term epistaxis is applied to bleeding from the nose; melæna, to bleeding from the bowels; hæmatemesis, to bleeding from the stomach; hæmaturia, to bleeding from the urinary passages; menorrhagia, to bleeding from the uterus.

DROPSY.

Dropsy is due to accumulation of serous fluid in the serous cavities, and in the interstices of the tissues of the body. More or less generally diffused dropsy of the integument is called anasarca. When localized, dropsy is called œdema. Ascites denotes dropsy of the peritoneal cavity; hydrothorax, of the pleural cavity; hydro-pericardium, of the pericardial cavity; hydrocephalus, of the ventricles of the cerebrum. Dropsical effusion is a colourless or straw-coloured, alkaline or neutral, clear or turbid watery fluid, having a specific gravity of 1010 to 1015. It holds albumen, salts, and extractives in solution. One of the chief pathological causes of dropsy is increased blood-pressure, which accompanies the several forms of congestion, especially that due to mechanical interference with the return of blood by the veins.

The pressure of the blood may also be increased independently of congestion, by obstruction to its flow through the capillaries, traceable to alteration of the relationship which exists between the blood in the capillaries and tissues around. Such a condition often accompanies chronic renal disease.

Dropsy may be local or general.

Local dropsy is frequently due to mechanical obstruction

of the veins, as in ascites from cirrhosis of the liver. It also occurs as the result of inflammation, and more rarely of lymphatic obstructions.

General dropsy is active or passive.

Active general dropsy is abrupt in its onset, and may result from a chill or other causes. It is widely distributed, and is frequently associated with febrile symptoms.

Passive general dropsy may be due to heart or renal disease, or anæmia.

HYPERTROPHY.

By hypertrophy we mean a quantitative overgrowth of an organ or tissue. The increase is either due to an increase in size of the individual tissue elements, or to an increase of their number. In the former case the hypertrophy is termed simple; in the latter it is termed numerical hypertrophy.

Hypertrophy is accompanied by increase in size and weight, and sometimes also by change of shape of the organ. Hypertrophy is regarded as the anatomical expression of increased function. In muscle, for example, where it is most commonly met with, the increase of work performed is followed by increase in size of those upon which the strain is thrown.

Since it helps to maintain the balance between the work to be done and the agent performing it, this hypertrophy is termed '**compensatory**.'

ATROPHY.

Atrophy is a diminution of the substance of an organ or tissue; it is thus the reverse of hypertrophy.

Atrophy is either simple or numerical, according as it is characterized by diminution of the actual size of the tissue elements, or by a diminution of their number.

These two varieties are generally combined.

Atrophy is accompanied by impaired function, which is frequently still further impaired by its association with degeneration of the tissue.

Atrophy is local or general.

Local atrophy may be due to—

1. Imperfect blood supply.
2. Impaired nutritive activity.
3. Excessive functional activity, leading to exhaustion.
4. Inflammation.
5. Nervous disturbance.

General atrophy is usually simple, affecting firstly the adipose tissue, then the muscles and viscera, and lastly the tissues of the nervous system.

It may be due to—

1. Deficient supply of nutriment.
2. Excessive waste; as in exhausting diseases.
3. Impairment of nutritive activity.

DEGENERATIONS.

Degeneration, unlike atrophy, is characterized by qualitative rather than by quantitative changes in the tissues and organs.

Two kinds of degeneration are distinguished.

In the one the albuminoid constituents are directly metamorphosed into some new material of lower type; whereas in the other the new material is deposited in the tissues from the blood.

The first kind is termed **Metamorphosis**; the second is termed **Infiltration**.

In both there is impaired function of the parts involved; but this is much more marked in the metamorphoses.

THE METAMORPHOSES.—There are three varieties; viz., fatty, mucoid, and colloid degeneration.

Fatty Degeneration.—This variety of degeneration is characterized by the direct conversion of the albuminoid substance, of cells or fibres, into fat. The cells or fibres enlarge at first and then break up, and the fat is thus distributed in the tissues. Caseation is a modified form of fatty degeneration.

The common causes of fatty degeneration are senile decay, deficient blood-supply, diminished vitality, congestion and inflammation.

Mucoid Degeneration.—In this form the albuminous intercellular substance, and far more rarely the cells, become liquefied and converted into a substance called mucin. Mucin is precipitated by acetic acid, and contains no sulphur, thus differing from albumen.

Colloid Degeneration.—In this form the albuminous contents of the cells (and not the intercellular matter) are converted into a jelly-like material called colloid, which differs from mucin in containing sulphur, and in not being precipitated by acetic acid.

THE INFILTRATIONS.—There are four varieties of infiltration; viz., fatty, lardaceous, calcareous, and pigmentary infiltration.

Fatty Infiltration.—In this form fat is deposited within the cells as distinct globules, which increase in size and tend to coalesce.

The protoplasmic cell contents are at first merely pushed aside, but they subsequently atrophy. As an example of fatty infiltration may be mentioned the conversion of connective into adipose tissue. It is due to the presence of fat in blood, in greater quantity than can be consumed by the economy.

This in turn may be due on the one hand to excess of

food, combined with insufficient exercise and fresh air; or on the other, to increased absorption of fat from rapid wasting of fatty tissues elsewhere.

Albuminoid, or Lardaceous, or Amyloid Degeneration.—In this form the material which infiltrates the tissues and elements is a peculiar homogeneous, translucent substance called lardacein. It impairs the vitality and functions of the tissues, and alters their appearance.

Lardacein is essentially a modification of albumen, with a deficiency of potash and phosphoric acid, but with an excess of soda and hydrochloric acid. When treated with a weak solution of iodine, lardacein exhibits a characteristic brown mahogany colour; but some other forms of altered albumen also exhibit a similar colour when similarly tested.

Methylaniline violet stains lardaceous substance violet red, the healthy tissue being coloured indigo blue.

Lardaceous degeneration is almost always of secondary origin, occurring as a sequel of some other disease; for example, prolonged suppuration, or marasmus. A case in which it followed strangles is recorded by Williams. Lardaceous degeneration first appears in the muscular coats of the small arteries, infiltrating the muscle fibre-cells and adjacent coats of the interior, and eventually affecting the other coats. Thence it invades the cells and intercellular substance of the surrounding parts. The affected tissue elements at first enlarge, become smooth and rounded, lose their nuclei, and tend to merge imperceptibly with adjacent cells similarly infiltrated.

A mass of homogeneous glistening material is thus formed, which is scantily supplied with blood, partly owing to the pressure exerted on the vessels by the swollen tissue, and partly to the narrowing of the channels of the small arteries by infiltration of their walls.

Lardaceous degeneration is especially liable to affect the liver, spleen, kidneys, intestines, and lymphatic glands.

Organs in which lardaceous degeneration is at all advanced are usually considerably increased in size, and their absolute weight and specific gravity are also increased. Their surface is smooth, and their capsule tense. Their consistence is firm and elastic. On section they present a peculiar homogeneous glistening transparent appearance, somewhat resembling wax or glue. Owing to diminution of the calibre of the blood-vessels, and to the pressure exerted by the new material, they contain but little blood, and hence are always pale on section.

Calcareous Infiltration consists in the deposition of calcareous particles, generally between, but partly also within the elements of the tissues. The particles are composed of salts of calcium and magnesium. This form of infiltration occurs either as the result of an excess of earthy salts in the blood, as in osteomalacia or extensive caries, or as the result of impaired nutrition of tissues, owing to which earthy matter normally held in the blood in solution is deposited in them. Of the latter arterial calcification will serve as an example.

Pigmentary Infiltration.—Here pigmentary particles are deposited in the tissue elements, just as fat or calcareous particles are deposited in other degenerations. This form of deposit has been noticed in the lungs of horses.

FEVER.

Fever is an abnormal condition of the system, characterized by undue elevation of temperature. The mean temperature of the horse, taken in the mouth and rectum, is estimated at 99° to 102° F. The average temperature of a large number of records of blood-horses, under similar conditions, taken by us in the morning, was 100·5° F.

The external parts of the body in the healthy condition

are generally speaking lower in proportion to their distance from the centre of the circulation, and are liable to much variation, according to the state of the atmosphere.

The temperature of the body represents the balance between the production and loss of heat, both of which factors are under the control of the nervous system, and are brought into constant relation with each other by circulation of the blood. In fever this equilibrium is disturbed, and the temperature rises.

If the temperature exceeds 106° F. it is called hyperpyrectic; if it exceeds 104° it is high, and if below 103° it is slightly febrile.

An elevation of temperature may result from diminution of heat, loss, or from an increased production of heat, or from both combined.

Fever, however, is in all probability mainly due to increased production of heat, as is evidenced by the rapid emaciation and the increased elimination of products of oxidation of tissues, viz., urea, uric acid, carbonic acid, etc.

In fever, other conditions also are associated with elevation of temperature:

The **skin** is generally hot, and dry. In some cases it is bathed in perspiration.

Restlessness and delirium, are common **nervous** symptoms.

The febrile condition is frequently ushered in by a **chill** or **rigor**, due to the excessive and spasmodic contraction of the cutaneous arteries. The skin is therefore either generally, or in certain parts relatively, or actually cold.

The **pulse and respiration** are quickened in proportion to the rise of temperature, and the character of the pulse is modified. In the normal condition the pulse varies in number from 36–40, and the respirations are about 8 per minute.

Digestive disturbance is indicated by the dry mouth, coated tongue, thirst, anorexia, and constipation.

The Urine is scanty, more highly coloured, of higher specific gravity, and its solid constituents are both relatively and absolutely increased. In some instances, however—in acute pneumonia, for example—the chlorides are said to be diminished and sometimes absent.

General Emaciation takes place in proportion to the intensity and duration of the fever.

Varieties of Fever are based upon (1) the course and character :

A fever is said to be **continuous** when it persists for some time, and with diurnal variations not exceeding the normal limits. It is **remittent** when the diurnal variations are exaggerated, but the minimum does not reach the normal. It is **intermittent** when the diurnal variation is exaggerated and the minimum does reach the normal. It is **relapsing** when the fever is paroxysmal, each paroxysm being separated by intervals of one or more days, during which the temperature is normal.

Varieties of Fever are also based upon the (2) severity and character of the febrile symptoms as a whole :

Fever is called **simple** when it occurs in a mild or trifling form. It is called **inflammatory** when it is associated with acute local inflammation ; **specific** when it depends upon the introduction of a specific poison in the blood (*vide* 'Inflammation'); **Malignant** when it is very severe and of an exceptionally fatal type.

Asthenic or **Adynamic** Fever is characterized by symptoms of great weakness and prostration, by comparatively slight rise of temperature, and by the skin being covered with cold clammy sweat.

The **Typhoid State** denotes a low febrile condition, in which the mouth and tongue become dry and the breath

becomes fetid. There is great frequency, feebleness, and sometimes irregularity of pulse; liability to passive congestion of dependent parts; low delirium, and coma.

It is supposed to be due to defective elimination and accumulation in the blood of the waste products of nitrogenous tissues.

Hectic Fever is of an intermittent or remittent type. It occurs in connection with persistent purulent discharges, comes on insidiously, and is of long duration. It is associated with marked progressive emaciation.

Termination and Stages of Fever.—The stages of fever are termed the 'initial stage,' the 'fastigial stage,' or 'acme,' and stage of 'defervescence,' or 'decline.'

Fever may terminate in death or recovery. In the event of a favourable issue, the temperature falls suddenly by *crisis*, or gradually by *lysis*. It may fall by a combination of these two methods, as, for example, in febricula, when it sometimes first falls several degrees by *crisis*, and then gradually reaches the normal by *lysis*.

COLLAPSE AND SYNCOPE.

Collapse and syncope are characterized by functional depression, and may result from a variety of causes. They may be due to some specific poison in the system; to high fever; to suppression of certain secretions; to the action of extraneous poisons, as, for example, antimony and arsenic; to excessive pain; to rupture or perforation of internal organs; to violent injuries, and to hæmorrhage, and other causes.

In Collapse the temperature usually falls; the skin is cold, and the respirations are slow and shallow. The pulse is small and feeble, sometimes rapid, sometimes very slow. There is extreme debility. The animal may be sensible, or there may be partial or complete insensibility.

Syncope differs from collapse mainly in the suddenness of the symptoms and in their shorter duration.

DEATH.

Death may begin at the heart, lungs, or brain. When the heart's action suddenly and completely ceases, death is said to occur from syncope. When its action more gradually ceases, being accompanied by a gradual failure in the general vitality, death is said to occur from asthenia.

When death is due to interference with respiration, it is said to take place from asphyxia.

Death from coma begins at the brain, and is characterized by stupor and insensibility.

Finally, death is said to occur from necrœmia when it is due to decomposition of the blood. In this case the heart ceases because the blood is no longer able to afford the necessary stimulus. As examples of this mode of death may be mentioned that occurring in purpura hæmorrhagica, scarlatina, and anthrax, and several other diseases.

CHAPTER III.

THE VEGETABLE PARASITES.

For many years scientific men have been endeavouring to discover the nature of the so-called contagion of the acute specific fevers. Now, each one of these fevers runs a more or less definite course, and presents special characteristics of its own by which it is recognised. The poison of each multiplies in a most marvellous degree, and one diseased animal may spread the fever among countless numbers by the agencies of contagion and infection.

Even so long ago as the Great Plague of London, the belief was expressed that the pestilence was probably due to some living organism, which entered the blood of man and multiplied there and that the virus was capable of passing from him, through the medium of the air or by actual contact, to others. But in those days men had not the means at hand necessary for the discovery of such living parasites.

That the poison could not be gaseous or liquid was soon obvious, and hence it was regarded as a solid in a state of fine division, and has been proved to be insoluble in fluids. These facts, as well as its remarkable power of multiplication, seemed to indicate that the contagion was some living organism; hence the origin of the germ theory of disease.

In 1838 Bassi and Audouin discovered the fungous nature

of the muscardine disease of silkworms; and in 1840 Henle expressed a belief that living organisms, probably vegetable in nature, were the causes of the acute specific fevers.

Long before Schwann and Cagniard de Latour had discovered that yeast consisted of cells, probably vegetable in nature, which multiplied by gemmation, a close parallel was noticed between the infective diseases and the processes of fermentation.

The germ theory started by Astier, Schwann, and Cagniard de Latour, and perfected by Pasteur is accepted by most scientists of the present day, and all the processes generally known as fermentation and putrefaction are looked upon as due to the action of vegetable organisms.

Now various micro-organisms have been demonstrated in the blood and tissues of animals suffering from various infective disorders, of which group the acute specific fevers form a division.

These organisms are of a vegetable nature, and as they possess neither root, stem, nor leaves, are referred to the Thallophyta. They have no chlorophyll, or green colouring matter, and they thus form a group of the class of Fungi.

There are three kinds of pathological fungi:

(1) **The Bacteria, or Schizomycetes,** *e.g.*, **Bacillus Anthracis.**

(2) **The Yeasts, or Blastomycetes,** *e.g.*, **Oidium Albicans.**

(3) **The Moulds, or Hyphomycetes,** *e.g.*, **Tinea Tonsurans.**

The Bacteria, besides causing putrefaction and several of the fermentations, include *almost all* the organisms which are believed to cause the infective diseases.

THE BACTERIA OR SCHIZOMYCETES are unicellular, strongly refractile, very minute organisms. They vary in shape, and appear to be structureless, though they may possess a cell-membrane allied to cellulose. They may be

round, oval, rod-shaped; straight, wavy, or in the form of a corkscrew. They are never branched, and they multiply by transverse division, which in the rod-shaped forms occurs at right angles to the long axis only, but in the round forms may take place in two directions at right angles to each other. In some of the Bacilli, spore formation has been observed to alternate with multiplication by the above process of fission. This spore formation is sometimes preceded by the growth of the rods into long filaments. The new cells thus formed by fission may separate or may remain united to each other, end to end, forming chains, or lying side by side in more or less spherical colonies, they are frequently bound together by a viscid intercellular matter—zoogloœa—formed of mycoprotein or swollen cell-membrane.

Bacteria multiply at an enormous rate, and Cohn estimates that a single organism may give rise to over 16,000,000 in twenty-four hours.

Single round cells have only what is termed the 'Brownian' movement; but chains and colonies do seem capable of locomotion (Ogsten). The red forms often have a mobile and a motionless stage; but some, *e.g.*, the Bacillus Anthracis, never move.

CONDITIONS OF LIFE: Food.—Each fungus must be supplied with the materials from which it can extract the elements necessary for growth. These elements, which will vary with each kind of organism, are carbon, hydrogen, nitrogen, sulphur, calcium, potassium, and magnesium. The first four are provided by carbo-hydrates (starchy or saccharine compounds) and albuminoids; the others by inorganic salts. Some Bacteria can assimilate nitrogen and carbon from much less complex bodies than albumen and carbo-hydrates, as is shown by the growth of putrefactive organisms in Cohn's fluid.

Very small variations in the composition of the food material may favour the development of one form rather than another.

As an example of this, it is noteworthy that Koch was unable to inoculate a field-mouse with organisms which always produced fatal septicæmia in a house-mouse. A similar difference may exist between two animals exposed to the action of a specific virus, when one of them is infected while the other escapes. Thus a very slight change in the system, or in part of it, may enable organisms to grow in it, though previously unable to do so.

Pasteur has divided Bacteria into ærobious and anærobious, according as they require for life free oxygen or not. The Bacillus Anthracis dies in the absence of free oxygen. Bacterium Termo and the yeast-plant can live with or without it. For Bacillus Septicæmia of rabbits, air is not only unnecessary but even fatal (Pasteur).

Temperature.—Each organism flourishes best at a special temperature. In some, as in Bacillus Tuberculosis, the range within which growth can take place is very limited; in others, the range is much greater.

The reproduction of Bacteria ceases at 5° C., and that of many at a much higher temperature, but death does not of necessity ensue.

Some are said to resist the greatest degrees of cold, and the spores of Bacillus Anthracis have been exposed to −140° C. without manifest injury.

Dry spores of Bacillus Anthracis are able to resist exposure to a temperature of 140° C. for over two hours.

Boiling, and even a much lower temperature than 100° C., will kill many forms. Boiling for one or two hours will destroy all non-spore-bearing organisms, but solutions containing spores will not be sterilized unless the exposure to this heat is continued for many hours.

Rest.—Some fungi flourish better in a still medium than in one constantly agitated. Bacillus Anthracis divides actively in the circulating blood, whereas other forms seem to settle before so doing.

Clinically, two great divisions of organisms are recognised: (1) The pathogenic, which can invade living tissue, and by their growth there almost always cause disease; (2) The non-pathogenic, which can only grow in dead tissue. Bacterium Termo, the cause of putrefaction, is the most important of these.

Infective diseases are divided, according to certain characteristics of the virus, into:

1. Contagious.—These are communicable from individual to individual, and are frequently epidemic. The virus runs its whole course of development in the body.

2. Miasmatic.—These are endemic diseases, of which malarial fever is an example. The poison which causes these diseases is developed outside the body.

3. Contagio-Miasmatic.—In these the poison has to undergo some change external to the body, and the diseases seem to be always derived indirectly from a previous case of the malady. As an example, cholera may be given.

4. Septic.—The poisons of these may be derived from many putrid infusions. When once thus started, the disease can be transmitted directly from individual to individual.

The Bacillus Anthracis is the best known of all the endo-parasitic fungi. As this germ is the cause of splenic fever of cattle, anthrax of the horse, and malignant pustule of man, it will be well to give a short outline of its distinguishing features and conditions of life, as well as of Pasteur's method of inoculating animals by the attenuated organism as a preventive against future invasion. In the blood from the enlarged spleen of animals dead of splenic fever are

found enormous numbers of rods, 5 to 20 μ long by about 1 μ broad, straight and motionless.

In a suitable culture-material, with a good supply of oxygen and a temperature between 15° and 42° C., the rods develop into very long filaments. In these filaments, round, highly refractile spores form at short and regular intervals. The filaments then break up, and the spores escape and may grow into bacilli. In living animals the rods multiply solely by division, and long filaments and spores are never found.

The rods are found in enormous quantities, especially in the spleen, lungs, liver, kidneys, mucous membrane of the intestines, and in various other parts.

Bacillus Anthracis is *always* present in splenic fever in enormous numbers. The bacilli may be separated and washed with distilled water, alcohol, ether, and dried; yet they still cause anthrax fever when inoculated into animals. Pure cultivations through fifty generations may be made with the same result. This germ always gives rise to anthrax fever, and never to any other. Therefore, in conclusion, *we recognise in this germ the direct cause of this malady.*

Now, M. Pasteur asserts that he has succeeded in modifying the micro-organism of anthrax by artificial cultivation, and that by inoculating animals with the attenuated virus he is able to cause a mild form of the disease, conferring a certain immunity against future attacks.

Pasteur's Method of Preparing Vaccine, or Attenuated Virus of Anthrax.—A drop of blood, on a glass rod, is taken from an animal in the last stage of anthrax. It is now placed in a suitable clear pabulum, such as fowl broth, previously rendered sterile by subjecting to a temperature of 115° C. The vessel containing the fluid is kept in pure air at a temperature of 42° to 43° C. The fluid gradually becomes cloudy, but no spores are developed.

Now, one drop of the fluid thus prepared, and in which the first degree of attenuation is produced, is put into another vessel containing fowl broth and placed under the same conditions as the first. This process is repeated until the requisite degree of attenuation is reached. The greater the intervals which elapse between each cultivation, and the greater the number of cultivations, the less virulent will the culture become.

The 'vaccine' may be injected into the subcutaneous tissue. Great care is required in obtaining the requisite degree of attenuation.

Large numbers of sheep and other animals have been thus inoculated by Pasteur, and the results of his labours attest the practical value of his investigations.

Quite recently M. Pasteur has notified that he has been able to confer immunity from hydrophobia by inoculating with the attenuated virus of this disease.

The Blastomycetes or Yeasts.—These are roundish or oval cells which sometimes form chains by joining end to end.

The yeasts multiply by gemmation, or when food is not abundant by the formation of spores in the interior of the cells. They never invade living tissues, and are of far less importance in the causation of disease than the Schizomycetes or Bacteria and the Hyphomycetes or Moulds. As an example of the group of yeasts may be mentioned the Oidium Albicans.

The Hyphomycetes or Moulds.—These consist of filaments called hyphæ, formed by a single row of cells placed end to end, growing by means of an apical cell which elongates and divides transversely.

The Thallus usually consists of numerous hyphæ, though it may be formed of one only. Several skin diseases are to be attributed to invasion by these vegetable parasites.

Tinea Tonsurans is due to the growth of Trichophyton Tonsurans, and Tinea Favus to Achorion Schlönleinii. Bursatee is similarly due to the growth of one of these moulds, as also is Actinomycosis and the 'Madura foot' of India. The mould causing Bursatee has been described by Mr. F. Smith, of the 12th Royal Lancers, in the *Veterinary Journal* for July, 1884.*

The Actinomyces, the vegetable fungus of Actinomycosis, is of recent discovery, though even so long ago as the year 1872 the late Mr. D. Gresswell expressed his belief that many of the indurations of a certain character met with in the tongues of oxen, and variously described as schirrhous and tubercular, were due to the invasion by a 'vegetable mould.' With this view, accordingly, he treated them with the injection of sulphurous acid, and sometimes of carbolic acid or tincture of iodine.

* Mr. Smith writes: 'The actual exciting cause of Bursatee is a mould fungus. I have found it in every fresh specimen of the sore that I have examined. . . . The filaments or hyphæ are branched not septate, $\frac{1}{2000}$ of an inch wide and so long that they cannot be followed as they pass in and out of the tissue. . . . I have lately produced typical Bursatee ulcers by inoculation with portions of the tumour, and am at present working this out, and endeavouring to produce the sore from the cultivated moulds.'

CHAPTER IV.

GENERAL DISEASES.

FEBRICULA, OR SIMPLE FEVER.

Definition.—Febricula is a simple fever characterized by rigors, elevation of temperature, quick pulse and respirations. It is a benign affection, and usually terminates in restoration to health, and unaccompanied by serious complications.

Etiology.—Among the causes of simple fever are: exposure to cold and wet, variations in temperature, fatigue, and irregular dieting.

Symptoms.—The first stage, *i.e.* the *attack*, is usually ushered in by a rigor or shivering fit, with rise of the internal temperature from 101° to 105° or more. The pulse is usually raised to 60 or 70, the respirations are somewhat accelerated, and there is loss of appetite. In the second stage, which follows in a few hours, the fever reaches its *acme*. The volume of the pulse is increased and the surface temperature rises, the bowels are costive, and the internal temperature remains elevated for several days. In the third stage, or *stage of decline*, or *defervescence*, as it is termed, the temperature usually falls several degrees by *crisis*, and then gradually falls to the normal by *lysis*.

Treatment.—Place the animal in a loose, well-ventilated box. In the early stage the surface temperature should be kept up by friction and by moderate clothing. Medicinally,

liquor ammoniæ acetatis, spiritus etheris nitrosi, and potassium nitrate or bicarbonate may be given three times daily. The diet should consist of bran mashes, linseed gruel, grass, carrots, etc.

STRANGLES.

Synonyms.—Febris pyrogenica; morbus glandulosus; gourme (French); colt-ill; druse, kropf (German).

Definition.—A specific febrile disorder, in which there are purulent accumulations in the connective-tissue of the submaxillary region and in the neighbouring glands, accompanied by inflammation of the upper air-passages.

Origin of the name Strangles.—This disease received its name from the great difficulty of respiration, accompanied by a trumpet-like sound, more marked in inspiration, these early signs resembling those of strangulation.

Geographical Distribution.—Widely spread over the whole world, but said to be absent in Africa and Arabia.

Varieties.—Benign and malignant strangles.

Prognosis.—The benign form seldom extends beyond a period of fourteen days, and nearly always terminates favourably. Malignant strangles may be of one or two months' duration, and the animal may die of various complications, viz., pyæmia, asphyxia, or peritonitis.

Contagium.—Strangles is, no doubt, both contagious and infectious, and may assume an epidemic form.

Etiology.—This disease is more prevalent in autumn and summer than in spring. Young horses, between the age of two and three, are especially liable to become affected, though it is not uncommon for aged horses to take it. The disease takes on the malignant form when the animals have been subjected to mal-hygienic conditions and to other debilitating influences.

Immunity.—An attack doubtless renders an animal less liable to future ones, but it is not absolutely protective.

Symptoms of the Benign Form.—The animal is dull and shows signs of mild catarrh. The disease is ushered in with simple continued fever. There is soreness of throat, slight cough, difficulty of swallowing, and disinclination to feed.

The pulse and respiratory movements are somewhat accelerated, and the temperature is elevated.

There is swelling in the neighbourhood of the throat, which may occupy the space between the rami of the lower jaw, or may be found on one side of the throat only, or may be more generally diffused. The swelling gradually enlarges, and ultimately pus may be detected by fluctuation. In some cases the glands and tissues in the region of the larynx are more seriously involved, and the symptoms become more severe. Œdema of the glottis, with implication of the other structures around, as well as general infiltration of the submucous tissue of the larynx, which may follow, cause difficulty of respiration and the production of the trumpet-like sound in inspiration and expiration.

This sound, which is most marked in inspiration, usually passes off under treatment. Sometimes this inflammatory condition is still more severe and persistent, and is a source of danger to life by suffocation.

Symptoms of the Malignant or Irregular Form of Strangles.—The fever is more severe and the tissues of the glands themselves become affected and suppurate. The parotid glands, and sometimes the inguinal, mesenteric, and tracheal glands become affected.

The fever sometimes lasts for a considerable period. When abscess is formed in the internal organs, the symptoms will vary in accordance with the organs involved, and the fever will be subject to intermissions and irregular

paroxysms. Exhaustion, acceleration of respiration, prostration, local dropsies, marked wasting of tissue, and occasionally irregular cardiac action follow.

Difficulty of respiration and cough usually occur, if the abscess be in connection with the mediastinum, and death may ensue from pus finding its way into the bronchi, or owing to the neighbouring organs becoming involved in the inflammation. If the mesenteric glands be affected, peritonitis may ensue and prove fatal.

Sequelæ of Strangles.—Roaring, or whistling, is sometimes left as a sequel of strangles. The most important sequelæ, however, are pyæmia and septicæmia, the former being accompanied by the formation of abscesses. The onset of pyæmia is accompanied by sudden increase in the febrile manifestations by regularly recurring paroxysms, irregular action of the heart, and accelerated respiration. Shortly, multiple abscesses form in various parts, especially in the limbs. The structures in connection with the joints may be invaded by these small abscesses. When the lung or heart or liver is invaded, there is but little hope of recovery, and pneumonic, cardiac, or enteric symptoms are developed.

Bacteria have been demonstrated in pyæmic lesions, and it is probable that when pyæmia results in cases of strangles, it is traceable to the fact that the animal has been under bad hygienic conditions during the primary malady.

Treatment of Strangles.—The animal should be placed under good sanitary conditions. In mild cases, such remedies as magnesium sulphate, sodium sulphate, potassium nitrate and bicarbonate will suffice. If laryngitis and tracheitis be present, camphor, ipecacuanha, and potassium nitrate may be administered. Inhalations of carbolised steam are of great value. Counter-irritants may be applied to the throat: for this purpose we may use ammoniacal liniments

or a smart cantharides blister. If these do not give relief, poultices may be applied to the throat.

Blister or poultice any enlargements, and when fluctuation is detected, open with the lancet, keeping the parts as clean as possible with an antiseptic solution.

The diet should at first be laxative, consisting of mashes, roots, grass, linseed gruel, etc. When the fever defervesces, tonics and stimulants and more liberal diet may be given.

Tracheotomy should be avoided, if possible, but if there is danger of asphyxia, this operation should not be delayed.

Method of performing Tracheotomy.—Elevate the head and make a longitudinal incision, about two inches long, in the mesial line of that part of the trachea least covered over with muscular or other tissue. This spot will be found to be about the junction of the upper and middle third of the trachea. Two or more rings may be divided transversely, or a circular portion may be excised. In the latter case a needle armed with a ligature is passed through a ring of the trachea, and then a circular portion is removed with the knife. The tracheotomy tube is now inserted, and is left as long as the difficulty of breathing continues. In malignant strangles, such antiseptic medicines as sodium salicylate, salicine, or sodium sulphite, are well worthy of trial.

INFLUENZA.

Synomyms.—Distemper; epidemic catarrh; the epidemic; epizootic catarrhal fever; panzootic catarrhal fever; febris catarrhalis; courbature, morfondure.

Definition.—Is a specific febrile disease, usually involving the upper air-passages, and subject to a variety of complications. It generally appears as an epizootic, and its contagiousness, though affirmed by many, is still denied by some.

Geographical Distribution and Historical Review.—

The first recorded outbreak of this malady occurred at Seville, in the beginning of the 14th century. In the years 1688 and 1693, severe epidemics occurred throughout Europe. In these epidemics horses were first affected, and then followed similar fevers in man. The last great outbreaks in this country were in the years 1863, 1864, and 1871 and 1872. The disease is widely distributed over the old and new worlds.

Varieties and Complicated Forms.—(1) The usual simple catarrhal form; (2) Complicated forms are—

A. The thoracic form.
B. The abdominal form.
C. The rheumatic form.

Prognosis.—Usually favourable.

Sequelæ.—If the pericardium be affected, pericardial adhesions may result, and if the heart be affected valve lesions may remain.

The upper air-passages may be so altered as to cause abnormal sounds, as, for example, roaring.

Etiology.—This disease appears in all climes and at all periods of the year, but is more prevalent in spring and autumn. It affects alike animals of all ages, breeds, and sex. It is not proved to be influenced by geological strata, climatic or astronomical causes. Animals are predisposed to attack by bad sanitary conditions, *e.g.*, by overcrowded stables and defective ventilation.

General Symptoms of Influenza.—Great prostration, with catarrhal inflammation of the respiratory, and sometimes of the other, mucous membranes, accompanied by pyrexial symptoms, characterize all the varieties of influenza.

Symptoms of the Uncomplicated Catarrhal Form.—In many cases the constitutional symptoms are but slightly marked. The legs and ears are usually cold, and there is a short irritable cough and sneezing. The throat is some-

times swollen and tender to the touch. The pulse is feeble and frequent, and the thermometer indicates a variable elevation of temperature. In a few days the temperature may rise to 105° or 106°; the pulse may reach 60—80 beats per minute, and the respirations become accelerated. The other symptoms also increase in severity: the cough becomes deeper and deeper; the throat becomes sore, and difficulty of swallowing follows in consequence.

The debility and prostration becomes more intense, and thirst is marked. The nasal mucous membrane, at first dry, becomes moistened, and there is a discharge of mucus. The bowels are sluggish, the fæces become hard and coated with mucus, and the urine is scanty and high-coloured, often containing albumen, or excess of urea and hippuric acid or of urates.

In about a week the symptoms gradually abate. The nasal discharge increases in amount, becoming thicker and purulent. The cough is moister, and the temperature falls by lysis, and convalescence is established in about fourteen days from the onset of the fever.

Sometimes, however, recovery is retarded, and the pyrexial symptoms recur. This is owing to the implication of the glandular structure around the throat, and of the neighbouring connective tissue, terminating in suppuration in the submaxillary and parotid glands, or in the surrounding tissue.

A Special form of Catarrhal Influenza, known by the names of 'pink eye,' 'pneumo-enteric fever,' 'epizootic cellulitis,' has of late been very prevalent. By some it has been regarded as a distinct disease, but it is probably only a modified form of simple influenza. In this form, the conjunctiva is of a pinkish colour, and in many cases the sub-conjunctival tissue is infiltrated with hœmorrhagic exudation. The constitutional symptoms are sometimes

very severe, and the temperature may rise to 105° or 106°, or even to 107°.

Sometimes severe pneumonic symptoms develop, when large portions of one or both lungs become implicated. Violent diarrhœa, colicky pains and other enteric symptoms, may supervene also. In some cases there is total loss of power in the hind-quarters, and sometimes, though rarely, cerebral symptoms may be manifested.

When occurring in strong horses, pneumo-enteric fever is usually a mild affection, but when attacking animals in a low condition it is more serious. The more the internal organs, as the lungs, intestines, and heart, are involved, so much more dangerous is the malady.

The sudden death which sometimes occurs has been attributed to the formation of thrombi in the cavities of the heart, and also to actual cardiac lesions.

Symptoms of the Complicated Forms—

A. Thoracic Form.—In this form the respiratory organs especially, or the cardiac organs, or both, may become affected, more commonly the former. Mr. Robertson divides the thoracic forms of influenza into (*a*) capillary bronchitis, (*b*) capillary bronchitis with cardiac disease, and (*c*) pleuro-pneumonia. All these forms may be detected by careful auscultation, and in all the pulse is frequent and feeble, and in cardiac disease of irregular rhythm.

The pneumonia of influenza is characterized by the soft dull cough, acceleration of pulse, coldness of the extremities and of the surface of the body. The pneumonia, being of the asthenic type, may terminate in gangrene and disintegration of the lung-tissue.

B. Abdominal Form.—This form is called 'bilious fever' by some, 'typhoid fever' by others. It is, however, in reality to be regarded as a complicated form of influenza.

In this variety the symptoms of gastro-enteric irritation

follow upon the catarrhal symptoms. The visible mucous membranes are yellowish in colour, the bowels are constipated, the fever is quickened, and there is great prostration; the pulse is remittent, and the temperature varies from 101° or 102° to 104°.

C. Rheumatic Form.—This form usually manifests itself towards the close of the ordinary symptoms of catarrh. It may be diagnosed by the pain in the joints, which crack when moved, and after a time swell.

Although the joints are especially liable to be affected, the muscles and the flexor tendons below the knee and hock are also sometimes involved. The swellings, like those of rheumatic fever, are metastatic. Cardiac complication may appear in this form of influenza.

Immunity.—One attack of influenza does not secure immunity from a second or even a third.

Morbid Anatomy of Influenza.—There is inflammation of the mucous membrane of the respiratory tract, extending into the minute bronchi, and pulmonary lobules are thus often found collapsed. In the various complications we find corresponding pathological lesions. In some large towns, patches of ecchymosis have been noted in the stomach and fore part of the alimentary canal.

The blood is dark in colour, and contains micrococci.

Treatment.—The animal should be placed under good hygienic conditions, and should be kept warm with rugs. Rest is in all cases essential.

The diet should at first be laxative, and consist of mashes, linseed gruel, roots, etc.

Medicinally, nitrate of potassium may be given in the water. Also liquor ammoniæ acetatis and spirit of nitric ether may be given in the form of a draught twice or three times daily. Sulphite of sodium in two drachm doses, or sulpho-carbolate of sodium in one drachm doses, twice daily,

may be given in addition. If laryngitis come on and be serious, camphor and belladonna may be administered, and hot-water vapour, medicated or not, may be inhaled. Blisters or poultices also may be applied to the throat.

℥ When convalescence is established, vegetable tonics with ammonium carbonate, or full doses of sulphate of iron or other tonics, may be given.

If the lungs are involved, the application of hot-water blankets to the chest is called for, or a smart counter-irritant may be applied.

If the bowels are affected, doses of morphia and chloric ether should be given to alleviate the pain, and hot applications or soap liniment used externally. If the bowels are costive, oleum lini or magnesium sulphate may be administered. If the bowels continue unrelieved, give a gentle dose of calomel. In the rheumatic form, hot applications and stimulating liniments should be employed for the swellings, and doses of bicarbonate of potassium given internally. If the temperature be high, salicylate of sodium is indicated.

GLANDERS AND FARCY.—EQUINIA.

Synonyms.—Equinia apostimatos; malleus humidus; farcina equi; affection morveus; morva farcino (Italian).

Definition.—A specific, highly contagious fever, which, though especially affecting the equine race, is readily transmissible to man, sheep, goats, felines and rodents.

The mucous membrane of the nose, frontal sinuses, and larynx and trachea, are specifically affected. There may be inflammation of the lymphatic glands, vessels, and skin, and small round buds known as 'farcy buds' may form and develop into pustules.

Geographical Distribution and Historical Review.—Glanders is described by Aristotle and Vegetius, and we read of it as far back as the time of Constantine the Great.

It is said to be absent in Australia, and rare in India, except when imported. It is mostly a disease of temperate climes, and is well known in Norway and Java. The form of the malady usually met with in this country is chronic glanders.

Contagion.—The contagium is present in many of the secretions and in the blood of the diseased animal. It is present in the greatest virulence in the discharges from the ulcers and nose.

Inoculation.—The disease is developed when infected material is given either in the water, or the food, or in the form of a bolus. The disease may be communicated by inoculating an abraded surface of the skin, or by injecting the virus into the blood-vessels.

Incubation.—The period of incubation varies. Most writers state it at three to seven days. It is, however, probable that it may sometimes greatly exceed this limit.

Varieties.—Acute and chronic glanders; acute and chronic farcy.

Etiology.—It is difficult to ascertain in what way the contagium gains access to the system. It is almost certainly due to some definite materies morbi, which will probably prove to be some form of bacterium (*vide* Chap. III.).

Horses which have been subjected to debilitating causes, as bad sanitary conditions, overwork, improper food, are especially susceptible to this malady.

Symptoms of Acute Glanders.—The febrile symptoms are very marked. Rigors may be persistent. The temperature rises rapidly to 105°, and may even reach 107° or 108°. It remains elevated for a variable number of days. The pulse is rapid, feeble, and sometimes fluttering, and its volume is diminished. The respirations are accelerated; the urine, especially when the fever is declining, is sometimes pale, increased in amount, and albuminous. The

appetite is impaired, and there is marked debility and emaciation. The Schneiderian membrane varies in colour from a light to a dark brown, and the other visible mucous membranes are highly congested.

In a few days (three to six) the febrile symptoms abate, returning again after a variable period of remission, during which the characteristic local lesions are developed. The pituitary membrane, especially that of the nasal septum and of the alæ, becomes studded with small tubercular nodules arranged in groups, or more generally diffused over the surface of the mucous membrane. When, as sometimes occurs, the subjacent tissue of the latter is much infiltrated, the patches become more prominent.

The nodules vary in size from a small seed to a pea, and appear as projections on an elevated base of congested mucous membrane. They have a yellowish white centre, surrounded by a greyish zone, round which is again a reddish one.

In a few days the diffuse patches and nodules gradually soften, and ulceration commences in the central portions of the latter. Thus, as the epithelium is removed, there is produced a characteristic ulcer with irregularly excavated borders. The floor and edges of the ulcer may become studded with small nodules, and covered with prominent vascular granulations.

The ulcers gradually extend and coalesce with one another, forming larger ones, which eat their way into the underlying tissue, and even at times lead to necrosis of the nasal septum.

The above pathological changes also involve the larynx and the mucous membrane of the sinuses of the head. The discharge from the nose, which during the onset of the febrile symptoms was yellowish and somewhat viscid, becomes, as the nodules ulcerate, thick, gluey, and purulent.

As the ulcers extend, the secretion may become sanguineous, owing to erosion of the walls of the small vessels. The neighbouring lymphatics and lymphatic glands, of one or both sides (according as *one* or *both* nasal cavities is affected), become enlarged. Those of the intermaxillary space are especially liable to become involved, and though at first painful and tender, rarely suppurate, but become harder and less painful. The lymphatic vessels, and especially those from the nose and mouth to the glands in the intermaxillary space, become nodulated, and may ulcerate and discharge a purulent fluid.

The scalp, face, and the tissues round the mouth become œdematous and swollen. The affection of the lymphatics spreads to those of the extremities, and nodules form on them and gradually disintegrate. Owing to the changes above described, the breathing of the animal becomes snuffling and hoarse, and there is in many cases frequent and painful cough.

Pneumonic or Pulmonary Glanders.—This is a form or complication in which the lung becomes variously complicated, from the formation of glanderous tubercles in its substance.

It can be diagnosed by careful auscultation and percussion, and by the characteristic soft dry cough. There is extreme debility and anorexia. Dyspnœa is marked. Œdema of the extremities and cutaneous infiltration ensue, and the disease generally proves fatal before the lapse of three or four weeks.

Symptoms of Chronic Glanders.—Chronic glanders may continue for many months without obviously affecting the general health of the animal. It differs from the acute form of the disease in the facts that its local phenomena are more important and numerous, and that the constitutional symptoms are more trivial and variable. The chronic form,

differing from the acute form, is more liable to develop constitutional signs at the later, than during the earlier, stages of the disorder. Usually there is a discharge—never absent where nodules or ulcers are developed—from one or both nostrils. The discharge is at first like that of common catarrh, but it gradually becomes thicker, viscid, pasty, and mingled with greenish yellow purulent matter, and has a tendency to adhere round the nasal orifices. Pathological changes similar to those described under the acute form occur in the mucous membrane of the nose, and in addition, parts of the latter occasionally become denuded of epithelium, the place of which is taken by a soft granular substance.

There is great tendency for the lesions not to heal, but to progress, and spread to the cartilage and the bone. The ulcerated and denuded surfaces may heal by cicatrization, the cicatrices themselves being sometimes slightly depressed. Although the nasal ulcers may heal, yet the disease may still remain, and nodules be present in the lungs and elsewhere. Eventually, also, the nasal ulcers may again resume activity.

Even when the sores have cicatrised the power of infection is not lost.

The lymphatic glands in the intermaxillary space enlarge, and are somewhat painful, but become gradually indurated, fixed to the jaw, and distinctly nodulated.

Chronic glanders is liable suddenly to manifest acute symptoms, especially during the latter stage of the disease.

ACUTE FARCY.

Definition.—Farcy is a form of glanders in which the cutaneous connective-tissues and superficial lymphatics, generally or conjointly, are especially liable to be involved.

Cause.—Farcy occurs as the result of direct inoculation

with the discharges of farcy or glanders, or from infection, and is said by some to arise *de novo* as the result of debilitating influences and mal-hygienic conditions.

ymptoms.—The first symptoms are those of fever. Rigors set in, and elevation of temperature to 106° or even to 108° may occur, but this height is not often reached. There are local swellings, generally confined to the extremities. They manifest themselves by engorgement of a whole limb, and resemble the swellings of acute lymphangitis. The engorgement appears as a generally diffused swelling, involving the cutaneous and underlying tissue, and the limb is hot and painful, and there is marked lameness. As the general œdema subsides, specific nodules, 'farcy buds,' and an enlarged condition of the veins and lymphatics become more manifest. Sometimes the œdema is not so generally diffused, but occurs in localised circumscribed forms, on which the specific buds arise. The lymphatics reaching from these buds to the lymphatic glands, or reaching from one bud to another, become enlarged and corded. The nodules generally appear in the œdematous tissue somewhat suddenly, and are situated in the skin or the subcutaneous tissue, and may extend even to the muscles. They vary in size from a pea to a marble, and are not sharply differentiated from the surrounding tissue. In a few days the buds disintegrate and the sores are formed. These farcy ulcers, several of which may coalesce, are deep, with ragged edges, and tend to extend and discharge an abundant creamy fluid tinged with blood.

As in the ulcers of glanders above described, the floor and sides of these rodent sores of farcy are covered with rounded elevations resembling vascular granulation-tissue.

Distinct nodules, which soften and discharge a yellowish purulent fluid, develop in the painful, swollen, and corded lymphatic vessels. The ulcers thus formed have likewise a

tendency to coalesce. The swollen and nodulated lymphatic glands themselves, as in glanders, very rarely suppurate.

The fever, which is of either the remittent or the hectic type, is liable to exacerbations marked by rigors and localised perspirations, and emaciation and prostration are marked.

Not uncommonly, acute glanders is developed in these cases.

CHRONIC FARCY.

Chronic farcy differs from the acute form only in intensity and duration. It is a very common form of equinia, and is more amenable to treatment than the other manifestations. The special features are eminently local. Fever, when present, is more remittent than in the acute form, and the constitutional symptoms are not so severe. In this disease, circumscribed inflammatory nodules, which vary in size, are developed in connection with the skin and subjacent tissues. They afterwards soften, ulcerate, and discharge a purulent fluid, and the ulcers have little tendency to heal. The nodules are especially liable to invade the skin where it is thin and vascular, as over the facial, maxillary and laryngeal regions, and along the neck, thigh, forearm, and flank. The lymphatic vessels and glands are also affected as in acute farcy, but the vessels are less liable to become nodulated, and the infiltrated state may remain in an indolent condition and eventually disappear. The glands themselves are not so painful nor so much swollen and infiltrated, and suppuration follows more rarely than in glanders or acute farcy.

Morbid Anatomy, Diagnosis and Treatment of Equinia.

Morbid Anatomy. The glanderous nodules which were mentioned as being found in the pulmonary tissues are of the size of a millet-seed, and grow in the connective-tissue

of the lung, and are subject to fatty, caseous, and calcareous change. On section they are of a translucent pearly-grey colour, surrounded by a zone of inflamed tissue. In structure they are identical with those found in the nasal cavity; but the latter, not being imbedded deeply in tissue, more readily ulcerate and discharge.

There remain for consideration two forms of morbid change, which, though important, are not frequently met with, viz.:

1. **Tumours, or Abscesses.**—These are generally found on the more exposed parts of the body: the scapular region, sides, and haunches. They are quite distinct from the nodules above described, and are larger, more defined, and less prone to ulcerate. They bear a greater resemblance to ordinary cysts or abscesses than to the specific lesions of equinia; but in the contained fluid, in disposition to fill when emptied, and in the slowness of healing, they agree with the latter.

2. **Diffuse Infiltrations.**—These infiltrations are usually situated in the limbs in the neighbourhood of the joints, and usually accompany the pseudo-rheumatic symptoms. They exhibit a tendency to rapid change of situation, appearing in connection with some other joint, and in this they resemble the rheumatic inflammations.

Diagnosis.—The diagnosis of equinia is usually not attended with difficulty; but in some cases, especially in those of pulmonary glanders, it is not always easy to arrive at a definite conclusion.

Glanders is now of far less frequent occurrence in this country than it formerly was, and this is largely due to the adoption of measures based upon its undoubtedly infectious character, and the management of horses in accordance with this idea.

Treatment.—Animals affected with the various forms of equinia are, in the interests of the community, destroyed, in order to prevent further spread. The rules of the Conta-

gious Diseases (Animals) Act are to be carefully observed. The stables should be thoroughly disinfected with sulphurous oxide gas, or by means of chlorine generated by the action of sulphuric acid on calcium chloride or bleaching powder (chloride and hypochlorite of calcium). The walls of the stables are thoroughly washed and scraped, and may be cleansed with lime-wash containing one pint of carbolic acid in each bucketful. The harness and fittings and other articles which have been in contact with the animal should also be thoroughly cleaned with carbolised water.

Remedies.—The remedies which have been used in equinia are arsenic, iodine, cupric biniodide, potassium iodide, sodium sulphite, and many others.

Of all internal medicines, iodine is in all probability the most potent in the various forms of equinia. It has been given in acute glanders in man with success, and has also been administered in many cases of the acute form in the horse.

VARIOLA EQUINA.

Synonyms.—Horse-pox; constitutional grease; variole du cheval; pferdepocken.

Definition.—Variola equina is a mild specific fever, accompanied by the development of an eruption which passes through a papular, vesicular, and pustular stage, and is preceded by a period of incubation during which no abnormal phenomena are manifested.

Incubation varies from four to eight days.

Diagnosis.—This disease has been mistaken for glanders and farcy, and also for eczema and impetigo.

Contagium.—Is propagated by direct contact, and probably also by infection. It may be produced by artificial inoculation in the horse and other animals, but the susceptibility of the horse to the virus of variola is greater than that of cattle

and sheep. The virus obtained from the vesicles and that in the saliva and crusts is especially potent. Whether one attack secures immunity against future ones or not is uncertain.

Symptoms.—Two or three days before the eruption develops, there may be mild fever and signs of impaired health, but these preceding symptoms are in most cases unnoticeable. The eruption commences as indurated red papules, circular in shape, and shortly becoming depressed in their central portions, and encircled by a bright red areola. In a few days the papules become vesicular, and contain a clear fluid which soon becomes more opaque.

Lastly, a scab forms, which if accidentally detached, discharges a limpid fluid, and leaves a depression, which gradually fills by granulation.

If the scab be not detached by force, healing by cicatrization, with little or no suppuration, will be accomplished in fifteen to twenty days.

During the period of eruption, and even after desiccation, there is usually considerable swelling of the limbs and inferior surface of the abdomen.

The swelling may remain for several weeks after convalescence.

Sometimes the pustules become confluent, and large ulcerating surfaces are then produced, causing death of large portions of the skin and subcutaneous tissue.

Situation of Eruption.—The eruption is especially seen on the extremities, from the knees and hocks to the feet. The hind limbs especially are liable to be affected, and often swell.

Other seats of eruption are the muzzle, lips, nostrils, the mouth, sides of the tongue, and the nasal mucous membrane.

In these places the spots are smaller than those on the

extremities; and with their presence are associated salivation, impaired mastication, considerable fever, and swelling of the submaxillary glands.

The eruption may be distributed over the whole body. Complete restoration to health is often retarded by the development of a secondary eruption of spots, or by complications.

Prognosis.—Is usually favourable. The disease generally lasts about fifteen to twenty days from the time of infection to the desquamative process. The eruptive stages extend over a period of eight days of this time.

Treatment.—Attend to the hygienic conditions. Salines and fever medicines may be administered, and the diet should be laxative.

If the mouth is sore, gargle with carbolic acid solution (1 part in 150 of water), or with a weak solution of zinc-sulphate or sulphurous acid.

ERYSIPELAS.

Synonym.—St. Anthony's Fire.

Definition.—Erysipelas (ἐρύω, *I draw*, and πέλας, *near*), is a specific febrile disease, accompanied by inflammation of the skin, and—in most cases—the subcutaneous tissues, with an eruption, not developed in every case; much pain, heat, and swelling.

Varieties.—(1) Simple cutaneous; (2) Cellulo-cutaneous, or phlegmonous; and (3) Cellular erysipelas.

Etiology.—Erysipelas is more liable to attack the weak and debilitated, and is probably due to some specific poison.

When occurring in association with an injury, it is termed *traumatic*, and when appearing spontaneously it is termed *idiopathic*.

With the traumatic variety it is our purpose to deal in the manual of equine surgery. The cellulo-cutaneous, or phlegmonous, form is the one most frequently met with in the horse. It is rather malignant, and involves the skin, subcutaneous and other tissues. The simple cutaneous form is rare, and, when it does occur, is a mild affection.

Symptoms.—These are : (1) general, and (2) local.

1. General Symptoms.—The fever, when occurring in young and in strong horses, is usually of the sthenic type, while in the older and in the debilitated animals it is of the asthenic or adynamic type. In all except the mildest cases it tends to pass on to a low prostrating form. The fever is ushered in by a rigor ; the temperature is raised, and there is muscular pain. The mouth is hot, the tongue furred, the breath has an acrid smell, and the bowels are confined.

2. Local Symptoms of the Simple Form.—Consist in redness (not easily observable in the horse), roughness, and slight swelling of the skin, which pits on pressure, accompanied by the formation of vesicles and frequently by serous infiltration of the subcutaneous tissue. Desquamation follows.

Local Symptoms of the Phlegmonous Variety.—The effusion into the subcutaneous tissues is at first serous, and the swelling pits on pressure. With the progress of the disease the parts become harder, more brawny, and more painful; and the local tension of the skin is increased.

Numerous vesicles, or phlyctenæ, preceded and accompanied by serous exudation, may be developed. They do not occur in every case, but when formed are a characteristic feature of erysipelas.

In the severer forms the serum of the vesicles becomes bloody and albuminous.

The inflammatory action going on in the subcutaneous

tissue may terminate in suppuration, and the pus formed may burrow in the meshes of the connective-tissue. Thus large portions of skin and tissue may be destroyed. If, however, the inflammatory action is not excessive, the effusion is absorbed, and the vesicles speedily desiccate and form scales, which separate in time. In cases of a very severe type, sloughing of the tissues may spread to the more deeply-seated structures, and involve the muscles, ligaments, sometimes even leading to necrosis of the bones.

In some cases the pus may find its way into the joints, causing their disintegration, and producing constitutional disturbance so serious as to prove fatal.

The mucous membrane of the mouth and nose may also become affected. In phlegmonous erysipelas the fever is very severe. The pulse is quick and feeble; the respirations are hurried, and the pain is intense.

Prognosis.—The milder forms may terminate favourably by desquamation in ten to fourteen days.

The phlegmonous variety, in which the fever is of a typhoid character, is in most cases fatal.

The cellular form is very rarely seen in the horse.

Diagnosis.—Erysipelas may be confounded with scarlatina, acute farcy, and lymphangitis. Yet by carefully noting the symptoms, they are easily distinguished.

Treatment.—The patient should be placed in a well-ventilated loose box, and a mild purge of calomel or aloes, or both, may be given. We may then administer liquor ammoniæ acetatis, with camphor and nitric ether, three times daily. If the temperature be very high, salicylic acid may be tried, in full doses, twice or thrice daily. The tincture of perchloride of iron is recommended by some from the earliest onset of the disease. In the human subject this remedy is regarded as a specific in erysipelas.

As the disease progresses, stimulants become necessary;

and aromatic spirit of ammonia, in doses of 2 ounces, or whisky, in doses of 3 or 4 ounces, may be given three times daily. Quinine may also be given, and other vegetable tonics. Potassium chlorate may be dissolved in the water during the acme of the fever, if the thirst be great.

Local Treatment.—Foment for two hours, three times daily, with warm water. When exudation has taken place into the subdermal tissues, free scarification is recommended by some. If abscesses form, they should be freely incised, so as to allow of the escape of pus, and the parts should be dressed with carbolic solution (1 part in 40).

Raw surfaces, and unhealthy sores especially, should be kept thoroughly clean by antiseptic solutions.

CEREBRO-SPINAL FEVER.

Synonym.—Epizootic cerebro-spinal meningitis.

Definition.—Cerebro-spinal meningitis is an acute specific febrile disease, often assuming the form of an epizootic, and affecting in particular the coverings of the brain and spinal cord.

Etiology.—The cause of this malady is not known. It is especially prevalent in America, where dietetic errors are said by some to produce it.

It is most probably due to some specific germ.

Symptoms.—The symptoms usually come on suddenly. They begin with dizziness and vertigo, quickly followed by total loss of motor power, which is often so complete and sudden that the horse falls prostrate to the ground.

The pulse and respirations are accelerated; there is often hyperæsthesia, or increased nervous sensibility, especially at the anterior part of the animal, and clonic contraction of

the superior cervical and dorsal muscles, passing in some cases to opisthotonos.

In most cases, although the wild look of the eyes and injected conjunctivæ indicate that the cerebrum is affected, yet consciousness is not usually impaired. When, however, the cerebrum is more deeply affected, coma with slow pulse and stertorous breathing follow. The bowels are constipated; the urine is often suppressed, and there may be incontinence, especially in mares. Pressure over the spine causes pain. The temperature is variable. It may, or may not, be elevated, and is intermittent.

In mild cases, which may be met with at any time during, but usually occur towards the close, of an epizootic, the leading symptoms are not so quickly developed. There may be pulmonary complications in this fever, and when this occurs the danger is increased. Priapism in horses and œstrum in mares are of frequent occurrence.

Morbid Anatomy.—Pathological lesions are especially marked in the membranes of the brain and spinal cord, which are found highly congested. The fluid of the subarachnoid space is turbid, and increased in amount, and all the vessels of the brain and cord are often congested.

In many cases patches of ecchymoses are found on the dura mater.

Prognosis.—If the symptoms come on slowly, the prognosis is, as a rule, correspondingly less grave. The number of fatal cases is large, and so the prognosis should always be guarded. The mortality may be as low as 10 or reach as high as 70 per cent., or even higher.

At the beginning of an epizootic the percentage of fatal cases is said to be higher. Even when a case is apparently progressing favourably, a relapse may occur.

Treatment.—If practicable, it is advisable to sling the animal as early as possible. The extremities should be kept

warm. Cathartics, *e.g.* aloes, with or without salines, may be given. Belladonna and ergot have been found of much benefit. The former may be administered hypodermically in the form of sulphate of atropia.

During convalescence, vegetable tonics with iodine may be given, and if paralysis remain, doses of strychnine, iron and quinine should be administered. Electricity also may be tried. In cases of great prostration, quinine or cinchonine and alcoholic stimulants are useful.

Locally, apply ice-bags to the spine, or hot water, as recommended by Dr. Chapman.

The spine may also be rubbed with stimulating liniments.

The diet should be nourishing and laxative.

ANTHRAX.

Synonyms.—Splenic fever; charbon; charbone; malignant carbuncle; gloss-anthrax; carbuncular fever; miltz-brand (German); carbone (Italian); jaswa (Russian).

Historical Review.—Anthrax is mentioned in the Scriptural narrative as the 'blain,' which affected both man and beast. It is described by the Greek and Latin writers. The former termed the disease οἴδημα when occurring in animals, and ἄνθραξ when affecting man. The Latin writers termed the disease *sacra ignis* in animals, and *carbunculus* in man.

Anthrax, though now of rare occurrence among horses, frequently raged as a malignant epizooty throughout Europe, in past times. The seventeenth and eighteenth centuries were remarkable for the devastations made by many epizootic outbreaks of anthrax.

In 1617 it was of such a fatal type, that around Naples over 60,000 persons perished from partaking of the flesh of animals which had died of the disease.

In 1731-32 an epizooty of gloss-anthrax raged among the domesticated animals in the States of Central and South-Western Europe.

The years 1757, 1763, 1779, 1780, and 1800 were marked by a charbonous malady, which extended nearly all over France, and affected all the domesticated animals.

Gerlach, in 1845, first demonstrated the contagious character of the disease; and Pollender, in 1855, first demonstrated the vegetable rod-like organisms, or bacilli anthracis as they are termed, peculiar to the malady.

Geographical Distribution.—Anthrax is of universal distribution, no clime being exempt from its ravages. As an epidemic and epizooty among men and animals, it breaks out in Siberia, where it is termed the 'Siberian plague.' As 'Loodianah disease,' it is of frequent occurrence in Central Hindoostan. In Australia it is termed 'Cumberland disease.' It is also met with in North and South America, and Central and Southern Africa.

Definition.—Anthrax is an acute infective disease, of rapid development and extreme fatality; appearing usually in an enzootic form; affecting all animals, including birds and fishes, but more especially solipeds and ruminants, and characterized by the presence and rapid multiplication of the bacilli anthracis in the blood and tissues.

Etiology—Direct Causes of Anthrax.—The immediate cause of this disease is the entrance of the bacillus anthracis into the blood, and its development there.

These vegetable organisms may gain entrance into the system by direct inoculation, by the digestive tract, or by the air-passages.

Predisposing Causes.—Anthrax is especially prevalent in low-lying, swampy districts, where the soil is rich in organic matter and salines; these conditions being in the highest

degree favourable to the growth and development of these low forms of vegetable life.

The same influences also may act upon animals, rendering them more fitted for the reception of these germs, and we know, from many experiments, of what great significance the nature of the soil or pabulum is to the growth and multiplication of bacteria. We may mention three instances:

Fowls were once believed to possess an immunity against anthrax. Now, M. Pasteur showed, by immersing fowls in water and thus lowering their temperature, he was able to inoculate them with the disease, though when not thus immersed he was unable to induce the development of anthrax in them.

Again, rats, when fed on animal diet, resisted inoculation, while others, fed on bread, readily contracted the disease.

Chauveau was unable to inoculate Algerian sheep by the ordinary method, though virus from the same vessel readily induced the disease in other sheep.

These experiments illustrate clearly of what importance the nature of the soil is to the growth of the germs placed in it. Animals in which no other disease is present, and those in a plethoric condition, are especially liable to attack, and young animals, also, are more likely to contract the disease than older ones. And it is in those warm, low-lying, marshy districts, where food especially rich in nutritive value is grown, and where, in consequence, animals are best nourished, that they afford a more genial soil for the reception of these vegetable parasites.

Again, it has been shown in many cases in moist districts, where anthrax has been long endemic, that after thorough drainage the disease disappeared entirely.

A hot, stifling atmosphere, charged with electricity, is said also to favour the development of these germs.

Modes of Propagation of this Disease.—Many attacks

have been traced to the indiscriminate burial of diseased carcases. According to M. Pasteur, the spores of the bacillus are brought to the surface by earth-worms, even ten to twelve months after burial of diseased carcases; but this method of propagation is doubted by some.

Dogs, after feeding on diseased flesh, may bite sheep, and thus inoculate them with the disease.

Flies feeding on anthrax blood have been shown to absorb sufficient poison in their proboscides to give the disease to animals inoculated with the contents.

Anthrax may be spread by eating contaminated food, by water percolating the soil and carrying with it the germs of the disease. Knives uncleaned may also propagate the disease.

The germs are said to be more volatile in spring and summer, less so in autumn, and still less in winter.

Although anthrax has not been shown to be infectious, yet there is every probability that it is. The disease often first appears in districts where it may be enzootic, and then assumes an epizootic form.

Incubation.—Varies from a few hours to one, two, or even four days.

Symptoms of Anthrax, without Special Localization, or Anthrax Proper, or Anthrax Fever, or Anthrax Apoplectica.—This disease is rare in Great Britain. The symptoms appear suddenly. The horse has violent muscular tremors, perspires freely, and breathes with difficulty and irregularly. There is complete loss of control of the muscular movements, especially of the lumbar muscles, and those of the posterior limbs in particular. The animal staggers and dies convulsed. These acute symptoms, especially those of nervous complications, may, however, abate in two or three days, and finally end in death or recovery. However, the symptoms are not always so

rapidly developed. They may begin with small, weak, and dicrotous pulse, uneven surface temperature, and violent tumultuous action of the heart. The internal temperature is high, but generally falls before death; the conjunctival membrane is swollen and of a yellowish-red hue. There is disinclination to move, inco-ordination, and loss of power in the muscles of locomotion, and twitching of the muscles in various parts. Drowsiness and stupidity, with much prostration, are marked symptoms. There is a yellowish discharge from the nose, often tinged with blood, and the nasal membrane is often covered with blood extravasations.

The breath is fœtid and the superficial lymphatics swelled; abdominal pain, shown by uneasiness, pawing, and looking to the sides, is sometimes manifested. The fæces are fluid or blood-stained; the skin is harsh and dry, and in some cases it crepitates when pressed towards the loins or sides. The respiration becomes tumultuous and hurried the nostrils dilated, and the animal foams at the mouth. The temperature declines, the animal staggers at every step; convulsions, and delirium or coma ensue, and death closes the scene.

Symptoms of Anthrax with Localization.—Anthrax in horses is not so frequently marked by local symptoms as in the ox and sheep.

'Nevertheless, a critical eruption of so-called anthrax tumours is sometimes present in certain epizooties, in which horses, as well as other herbivorous animals, are involved' (Fleming).

At a certain stage in the febrile symptoms there is an external eruption in various regions. This consists in the development of 'tumours' or 'anthrax-pustules' in the interstices of the muscles, in the loose areolar tissue, and in the lymphatic glands. They are especially met with in the submaxillary space in the upper part of the throat, the

lower part of the neck behind the shoulders, on the back, and in the inguinal region.

They may appear as small rounded nodosities, about the size of a walnut, and having as it were a pedunculated base.

In other cases the tumours appear suddenly as large, soft, crepitating, undefined swellings, and invade many surrounding parts. As they rapidly extend they become cold and insensible, and vesicles or phlyctenæ appear on the surface. These vesicles, quickly bursting, discharge an irritant serous fluid. If the hand be passed over these swellings crepitation may be felt. This is due to gas from the decomposition of the tissues collecting in the connective-tissue, and thus rendering the part emphysematous. This is a special feature of the tumours of anthrax.

Sometimes the tumours appear without being preceded by any very marked symptoms, and their course is in all cases the more rapid the earlier they make their appearance. In two to eight hours they attain a large size, and the surrounding tissues mortify as they are invaded.

As the tumours are developed, the fever abates and the urgent symptoms vanish. When they have attained a certain proportion, general symptoms are developed, which vary according as the malady takes a favourable course or not.

In some rare cases the tissue of the tumours is suddenly absorbed, and abundant sweats and fetid diarrhœa ensue, and the animal recovers rapidly. In some cases the tumour disappears by resolution, but generally the tumours vanish, and coincidently the disease assumes all the characters of anthrax fever without local manifestations; the general symptoms reappear, and the horse dies in about twelve hours.

ANTHRACOID DISEASES.

There remain for consideration two forms of anthrax characterized by pathological lesions of special parts. These are glossanthrax and anthracoid angina.

Neither has as yet been demonstrated to be due to the presence of bacillus anthracis in the blood. Neither form is common, and both are generally found associated together. By some authors they are termed anthracoid diseases (ἀνθραξ and εἶδος, like).

Glossanthrax.—The local symptoms which are rapidly developed in glossanthrax probably slightly precede the general disturbance of the system. Glossanthrax is a rare disease, and is generally associated with anthracoid angina. There is an eruption of vesicles, or phlyctenæ, on the sides, dorsum, and sometimes on the frœnum of the tongue, and on the buccal membrane of the mouth and lips.

The vesicles vary in size from a nut to a hen's egg, and contain at first a clear yellowish serum, which gradually becomes of a brownish-black colour. They rapidly increase in size, and break, and the acrid contents are discharged, being mingled with the abundant flow of saliva.

The tongue itself is swollen and hard, hanging from the mouth, and is of dark bluish or black hue.

It is often lacerated by the teeth, and deglutition becomes difficult, and the animal is soon unable to swallow fluids.

When the vesicles have burst, they leave an unhealthy ulcerated surface, which often has a gangrenous appearance, and becomes coated over with a yellowish exudation. This is removed, and exposes the ulcerating sore.

The vesicles usually form a few hours before death, and the constitution, previously not affected, becomes seriously so, and the animal usually dies in twelve to twenty-four hours. The condition of the tongue is often associated with inflam-

mation and swelling of the throat and the structures around the throat—anthracoid angina.

Anthracoid Angina.—This is a disease rarely occurring unassociated with glossanthrax. It consists in infiltration of the glands and connective-tissue in the region of the throat, the swelling being hot and painful. The swelling proceeds rapidly, involving the structures of the head, and soon the glottis and pharynx become œdematous. Thus the breathing becomes difficult, and death results from asphyxia.

There is often a sanguineous discharge from the nose, accompanying the swelling around the throat and larynx.

THE LOODIANAH DISEASE.

This disease is the form of anthrax commonly seen in India, the name being derived from the fact that the malady assumes a very severe form in Loodianah, in India.

It is especially seen in low damp situations where the sanitary conditions are defective.

The symptoms differ very little from the anthrax fever occurring in Europe. The temperature may in some cases reach as high as 108° or 109° F.

The percentage of recoveries is small.

THE CAPE HORSE-SICKNESS,

or Paard-Zietke, is another form of anthrax which affects horses in the Cape of Good Hope and other parts of Southern Africa.

Immunity from Anthrax.—One attack does not secure immunity from a second.

Morbid Anatomy of Anthrax.—Animals which die of anthrax are especially prone to decompose rapidly.

The subcutaneous connective-tissue is found distended with a yellowish serosity and gas. The skin itself is stained.

The intestinal mucous membrane is softened, deeper in colour, and studded with rounded infiltrations. Peyer's patches are enlarged; the peritoneum presents blood extravasations, and the folds often contain a yellowish exudation. The mesenteric glands are swollen and infiltrated, and the veins all over the body contain dark blood.

The pleuræ are ecchymosed and stained, and the pericardium and endocardium are similarly affected. The walls of the heart are weak and flabby, and the pericardial sac contains fluid. The lungs may or may not be congested, and there is frothy mucus in the air-tubes, and the lining membrane is infiltrated. The spleen is enlarged, sometimes being treble the ordinary size, and is friable and full of black tarry-looking blood. The liver is abnormally friable. The pelvis of the kidney and the lining of the bladder are ecchymosed.

The brain is congested, and its membranes also. There is also effusion into the subarachnoid space. The spinal cord and its membranes are similarly affected. There is infiltration of the yellowish exudation into the retro-pharyngeal and laryngeal tissues.

In anthrax attended by local manifestations, we shall find carbuncular elevations, above described, and large, deep, yellow ulcerated cavities.

Blood.—There is decrease in the elements of fibrine; the white cells are increased in number; the red cells cohere in masses, and are shrunken.

Bacilli may be observed under the microscope as cylindrical rods 5-20 μ in length, and about 1 μ in breadth, straight, with slightly concave ends and motionless. The rods elongate into filaments which divide by fusion. Spores develop at 30° to 35° C., and give a beaded appearance.

If the temperature is lower than this, spores are not developed, nor are they formed if the temperature is above 40° to 47° C.

There is no development without oxygen.

(For further particulars of the Bacillus Anthracis, *vide* Chap. III.)

The post-mortem appearance of anthrax will vary much with the symptoms shown during life; and in cases of artificial inoculation bacilli will be found at the seat of the lesion.

Morbid Anatomy of Glossanthrax. — Pathological changes are especially found about the larynx and tongue, and consist in extensive infiltration of yellowish exudate and extravasation of blood in that vicinity. The tongue is swollen and of a dark purplish hue, and presents vesicles and sores left by the bursting of some of them.

The salivary and lymphatic glands are enlarged and infiltrated, and the glottis is œdematous.

Treatment of Anthrax.—Give a mild laxative, *e.g.*, aloes or salines, and also some antiseptic remedy, as carbolic acid, sulphite of sodium, or sulpho-carbolate of sodium.

If carbolic acid be selected, 30 minims may be dissolved in a pint of water, and given thrice daily. If sulphite of sodium be selected, it may be administered in large doses thrice daily in the acute stages of the disease.

Special symptoms, such as abdominal pain, will require special treatment in addition.

Mineral acids may also be given. Chlorine and other inhalations have been recommended. If there be much debility, alcohol may be administered, and tonics should be given in convalescence. The food should be as nourishing as possible, and should be given in a liquid form. If the animal is unable to swallow, it will be necessary to give it in the form of an enema.

Tumours, when formed, should be incised, and either cauterized or dressed with antiseptics.

Treatment of Glossanthrax.—If the vesicles appear on

the tongue, they should be opened and dressed with carbolic acid solution. The internal treatment is the same as above recommended for anthrax.

If the tongue is much swollen, we may scarify deeply, and treat with a solution of carbolic acid (2 per cent. solution).

Hot-water vapour when inhaled is of benefit. It may be medicated with advantage. The swellings should be fomented with hot water. In some cases where the swelling is producing suffocation, tracheotomy is necessary.

Prophylactic Measures in Anthrax.—Carefully examine into the food and water supply, and take special care that there be no escape of effete matter into the wells or ponds.

Ascertain if the food be mouldy or fermenting, as mouldy grains have been asserted to cause anthrax in some instances. (*Vide Veterinarian* April, 1878.)

The healthy animals should be isolated from the affected ones; and the stables in which the horse contracted the disease or died, and all implements, such as harness fittings, etc., should be thoroughly disinfected and cleansed. The carcases should be interred deeply, and the litter and manure should be burned (Fleming).[1]

[1] In an article in the *Veterinary Journal* of January, 1884, we have recorded a large number of cases of anthrax fever in horses and beasts treated by the administration of sulphite of sodium. We believe it to be of the greatest value in this disease, and of more potency than any other antiseptic, and we also believe it to be in a measure preventive. The late Mr. D. Gresswell, F.R.C.V.S., used this remedy in all cases of anthrax in the horse and beast coming under his care. ——

In the *British Medical Journal* of June 14, 1884, Mr. Baker, F.R.C.S., Surgeon to St. Bartholemew's Hospital, records a case of 'Malignant Pustule' in man treated by excision and the administration of sodium sulphite. The disease was contracted from handling infected bales of wool from China. The case terminated in complete recovery. Mr. Baker adds that he was induced to select this drug from a perusal of the cases recorded by us in the *Veterinary Journal* and the success attending its use.

PYÆMIA AND SEPTICÆMIA.

'These diseases result from the absorption and dissemination of substances derived usually from septic disease of some wound or inflammation' (Green).

By *septicæmia* we understand a form of septic disease unaccompanied by the development of secondary inflammations.

By *pyæmia* is meant a septic disease characterized by the presence of secondary or metastatic suppuration.

Nature, Pathology and Morbid Anatomy of Septicæmia and Pyæmia.—Koch has shown that by injecting five minims of blood or meat, in an early stage of putrefaction, under the skin of a house-mouse, the animal at once becomes restless, and its movements weak and uncertain.

The respirations become slower and irregular, and death occurs in about four to eight hours. No pathological lesions are found, and the blood taken from the animal and inoculated into others has no effect. The disease is therefore non-infective, and as it follows the absorption of putrid matter into the blood, and is not accompanied by secondary inflammation, it is therefore a septicæmia.

The effect of the poison is comparable with the results of the injection of a poisonous alkaloid. This form of septicæmia is termed '**septic intoxication.**'

Sanderson gives the following as signs of septic intoxication in animals:

'Restlessness, muscular twitching, and increasing weakness; profuse diarrhœa, the fæces being loose, whitish-grey, and later bloody. The temperature rises at first, but often falls before death. Respiration and heart's action gradually fail, and death may be preceded by cramps.

'The post-mortem changes found are:

'Blood dark; petechiæ beneath the pericardium, endocardium, and pleuræ. Intense staining of endocardium and lining of the large vessels, and often a little blood-tinged serum in the serous cavities, both soon after death, indicating destruction of red corpuscles even during life; intense congestion and ecchymosis, with shedding of the epithelium of the mucous membrane of the stomach and intestines; spleen swollen, soft and pulpy; liver often swollen and congested' (Green).

If now house-mice be inoculated with less quantity of poison, the effect on the system will also be lessened. If a drop or two only of the blood or meat infusion be injected under the skin, many of the animals will show no untoward symptom, while about a third of them will gradually sicken and die. The symptoms thus manifested are: Dulness of the eyes; slow respiration; great weakness and prostration; and death occurs gradually in forty to sixty hours after inoculation. After death there is slight œdema, sometimes absent, at the place of inoculation, and the spleen is much enlarged.

Now, again, if a house-mouse be inoculated with a *most minute portion* of the blood from one of the animals just dead, the disease will be produced in it, and death will ensue in less than two days.

Now, this disease thus engendered is a septicæmia. But it differs from the first-mentioned form of disease in being *intensely infective.*

The poison introduced gives rise to the disease by multiplying in the blood, and not by reason of its toxic properties, for it is in too small amount.

A period also elapses between the time of inoculation—the period of incubation—and the manifestation of symptoms, during which the germs multiply. This period may be about twenty-four hours.

This form of septicæmia is termed **septic infection.**

'The blood of animals which had died after injection of one to ten minims of putrid blood contained bacteria, cocci, and bacilli; but after inoculation it contained only bacilli' (Green).

There are thus two forms of pathological conditions under the heading Septicæmia, viz., Septic Intoxication and Septic Infection.

The former is due to absorption of chemical poison, manufactured in some putrefactive process going on external to the body. The latter is due to entry of specific germs into the blood, and to their multiplication there. The organisms in these cases probably act by producing poisonous substances in their growth, but these products are not irritant, and therefore secondary inflammations do not ensue. Fungi which occur in the septicæmia of one animal differ from those which occur in another.

In **pyæmia,** the absorption and dissemination of the poison gives rise not only to the general disease, but also causes the formation of secondary foci of inflammation, which are termed metastatic abscesses.

The clinical symptoms of pyæmia are well marked; the irregularity of the temperature being the most prominent feature. Pyæmia is complicated with more or less septic poisoning. The source of the infection in pyæmia is almost always a wound or inflammation, generally suppurating, the discharge being septic. In some cases, however, no wound is traceable. As in septicæmia, the poison gains access to the blood-current, which distributes it through the system.

The secondary abscesses of pyæmia are of two kinds; those which are preceded by infarction, and those in which we have no evidence of this antecedent occurrence.

The suppuration is probably due to the parasitic fungi irritating the tissues in which they are placed. In the

former kind of abscess the infarction is due to the lodgment of an infective clot in a terminal artery. The most frequent seats of these embolic abscesses, as they are termed, are the lungs especially, the liver, spleen, kidney, and brain, but they may occur in any vascular part of the system.

They vary in size, and are usually multiple, and may be very numerous.

The other kind of abscesses are diffuse suppurative inflammations in the subcutaneous and connective tissues, in the joints, and in the serous membranes. They are fairly often met with, and may or may not coexist with the embolic abscesses above mentioned.

Besides the secondary abscesses, there are other lesions found on post-mortem examination. The wound, if present, is in a sloughing condition, perhaps surrounded by diffuse infiltration of the tissues, and is offensive. There are extensive thrombi in the veins leading from the point of infection; these thrombi are undergoing infective puriform softening.

The blood is darker than in the natural condition, and not disposed to coagulate, and contains *excess of white blood-cells.* Congestion of the lungs is generally present; the spleen is large and pulpy; the liver and kidneys show granular degeneration.

Etiology of Pyæmia in the Horse.—Pyæmia is chiefly developed in connection with injuries and wounds, and more especially in connection with suppurative-action in bone.

It may accompany various diseases, notably the specific fevers.

Symptoms of Pyæmia in the Horse. — Rigors and localized perspirations. Sudden elevation of temperature, which rises and falls irregularly. It may rise to 106° or more, and, though variable, is always high.

Pneumonic symptoms are often developed from the

formation of abscesses in the lungs. Also other special symptoms, due to the formation of abscesses in particular organs, are frequently manifested. The skin may be jaundiced, and show petechial markings. In cases following wounds, the latter, a few days after infliction, show abnormal changes.

Infiltration, leading to œdema and gangrene of the surrounding connective-tissue, comes on, and the wound discharges an unhealthy ichorous matter, and gases accumulate in the subcutaneous tissue. When pyæmia accompanies the specific fevers, it usually first shows symptoms after the acme of the fever has been passed.

Prognosis of Pyæmia.—This disease is generally fatal.

Treatment.—Allow plenty of fresh air, and a liberal and nourishing diet.

The drugs which are specially recommended are—quinine, arsenic, salicine and salicylate of sodium, and iron. Alcoholic stimulants are, no doubt, of great value in the treatment of pyæmia.

Carbolic acid has also been recommended in moderate doses. If there be any wound, it should be carefully attended to, and well dressed with antiseptic lotions.

SURRA.

Surra is a disease widely spread in India. The name 'surra' signifies 'rotten,' and the disease is so termed from the great emaciation occurring in animals affected with it. Our knowledge of this malady is due to the members of the profession residing in India—especially to Mr. Griffith Evans, of the Army Veterinary Department.

Definition.—Surra is an enzootic disease, due to the presence of a special animal parasite in the blood, and characterized by progressive emaciation, dropsical swellings,

elevated temperature, and extravasation into the visible mucous membranes.

Symptoms. — The symptoms are somewhat variable. Fever is present; there is a yellowish discharge from the nose, and sometimes the submaxillary glands are enlarged, and may discharge.

The visible mucous membranes are yellow, and present petechiæ which are especially seen on the inner canthus of the eye, and in the female in the vagina. The appetite is generally good. The urine contains albumen, and is highly coloured; and there is gradual progressive emaciation and marked thirst.

Sometimes in the early stages there is loss of motor power in the limbs. Dropsical swellings are sometimes seen in the mare between the fore-legs, and in the horse in the sheath.

Etiology.—The disease is due to the presence of an animal parasite in the blood. It is capable of being propagated by subcutaneous or intravenous injection, or by introduction into the alimentary tract of blood infected with parasites. It is not known how the disease is spread, but probably the parasite gains access into the system through the drinking-water.

Prognosis.—Death usually occurs in seven to ten weeks, at the end of which time the animal may expire suddenly, or may become delirious and death ensue more slowly.

Morbid Anatomy.—The intestinal membrane is yellow, and shows small extravasations.

The peritoneal and pericardial sacs contain lymph.

If fresh blood be examined under the microscope, the parasites may be seen in groups or singly.

They have an oval-shaped body, separated from a spheroidal head by a neck, and the tail tapers into a long flagellum.

In the cervical and in the caudal region there is a papilla-like eminence.

The length of the parasites is about three or four times that of a white blood-cell. They are whitish in colour, and appear in the field to attach themselves to the red corpuscles. The white corpuscles are increased in number.

Treatment.—Turpentine, carbolic acid, and other antiseptics are worthy of trial.

MALADIE DU COIT.

Synonyms.—Syphilis du chéval; epizootic paralysis; contagious eczema; vérole.

Definition.—Is a specific contagious disease, characterized in the early stages by morbid lesions in the urino-genital apparatus.

Varieties.—Benign and Malignant.

Geographical Distribution. — The disease was seen in Russia in 1796. Since then it has occurred in parts of Africa, Egypt, and most European countries. There is no record of its occurrence in Spain, Belgium, and Great Britain.

Etiology.—The origin of the disease is obscure. It has not been proved to have any relationship with syphilis.

Symptoms of the Benign Form.—In the female this disease appears in one to fourteen days after copulation. The first symptoms are often so slight as not to attract attention. The symptoms are the same as those shown in œstrum; but in this disease the sexual desire is unsatiable. The vaginal mucous membrane is red, and discharges a muco-purulent fluid, which, slight at first, becomes thick and viscid and coagulates on the adjacent parts. Pustules about the size of millet seeds appear in the vagina and fossa navicularis. The pustules become ulcers, which rapidly heal and

are replaced by others. There is œdema of the abdomen and extremities, and some engorgement of the submaxillary glands.

The symptoms gradually disappear in two weeks to two months.

In the stallion, beyond swelling of the sheath, this form of the maladie du coït is unnoticeable.

The benign may pass into the malignant form in either sex.

Symptoms of the Malignant Form.—In the mare the symptoms begin in the same way as in the benign form, but soon become more pronounced.

The lips of the vulva alternately swell and diminish in size. The anus, mammary gland, and perinæum are swollen; the vaginal mucous membrane presents furrows, becomes more infiltrated, and presents blue and yellow patches, and pustules are formed on its surface. The discharge from the vagina is excoriating. Lenticular pustules may appear on the external surface of the lips of the vulva, on the perinæum, and inside the thighs. Scabs form over them and leave circular ulcers, which heal rapidly. The clitoris is swollen, and the discharge becomes reddish green and fetid. Urine is ejected frequently and in small quantities, and causes much irritation when passed.

Mares in foal usually abort at the third month, and if the foal is born it is dead or soon dies.

In the stallion the symptoms are very uncertain; the disease may remain latent for a very long time. The sheath is œdematous, and the swelling may extend in front and behind it. The testicles may or may not become swollen. The penis also may or may not be altered; sometimes purple spots are found, and the glans during erection may be very large. The urethral mucous membrane projects in a globular form from the meatus, and is crimson

or yellow. Urine is passed in a small jet, and with much pain.

Paraphymosis, the result of local swelling, is not uncommonly met with.

General Symptoms, common to both Sexes.—There is a muco-purulent discharge from the nose, and also a discharge of tears from the eyes. The visible mucous membranes are highly injected. The coat is dry, and the perspiration, when breaking out, is offensive. Pressure over the lumbar region causes pain, and the animal rests first on one hind-limb and then on the other. The lymphatic glands in the neighbourhood of the genital organs are engorged, and those in the submaxillary space are similarly affected. The animal is much debilitated, and walks with uncertain gait; the large joints are tumefied and painful; the haunch especially is affected. The pain causes the animal to lift its leg while at rest, with a sudden jerk.

The appetite is generally pretty good. Often one or both hind extremities becomes paralyzed, and the entire muscular system becomes atrophied, especially the hind-quarters and legs.

The nasal membrane becomes yellow, and the buccal membrane of purplish hue.

The mammary gland may inflame and suppurate. During the incubatory stage of this disease numerous cutaneous tumours form on different parts of the skin. They are small, of medium size, or large; the small ones appear and disappear suddenly; the larger ones develop in twenty-four hours, and disappear in from four to eight days.

Albumen is present in the urine.

Prognosis.—The disease usually lasts three or four months, but may extend over a period of three years.

Contagion.—The disease is readily transmissible by copulation. Mares take the disease more readily than stallions.

Morbid Anatomy.—The external lesions are always present. The sheath is infiltrated with serous exudation, giving it a yellowish, hard, homogeneous appearance when cut into. The penis, especially the glans, is also infiltrated, and the mucous membrane of the vesiculæ seminalis is red or violet coloured, and contains a purulent matter.

The testicles are sometimes enlarged, and contain yellow serosity; they may be atrophied. The uterus contains yellow or brown coloured muco-purulent matter; the kidneys are sometimes normal, but sometimes enlarged, and contain a white albuminous substance. The membrane of the bladder is thickened, ecchymosed, and of a reddish brown colour. The muscles and bones are friable; the coxo-pubic and femoral ligaments are red, thickened, softened, and may be ruptured.

The synovial fluid of the joints is abundant, cloudy, and dark-coloured. The cartilages are soft and yellow. The sub-glossal, sub-lumbar, mesenteric glands, and those near the genitals are enlarged, yellow or reddish, and often contain pus. The spinal cord, especially in the lumbar region, is sometimes softened. The sub-arachnoid fluid of the brain is increased, and according to some the brain itself is in a degenerate condition.

The sacro-sciatic nerves and lumbar plexus are infiltrated with serum.

The sinuses of the head often contain a yellow oily matter. The blood is much altered, and is fluid and deficient in fibrin.

Treatment.—M. Trelut gives fibrin, ʒxv., as an electuary, or in gruel as a drench, in the morning; also, turpentine, ʒv.—vii. ss., every second morning with the drench.

From ʒi.—ii. ss. of reduced iron, alternated with gr. xv.—xxx. of white arsenic, is given in addition. Food of the most nutritious kind is allowed. M. Trelut after-

wards substituted cooked horse-flesh for the fibrin. When paralysis set in, cantharidine liniments, followed by mustard poultices, were applied to the abdomen, and, when there was effusion the pointed firing-iron was applied, penetrating as deeply as possible.

The treatment is continued two or three months.

Local Treatment.—In stallions, when the disease is confined to the genital organs, castration has been performed.

In the mare, emollient injections, followed after by astringents, have been recommended.

PURPURA HÆMORRHAGICA.

Definition.—Is an eruptive, non-contagious, intermittent fever, characterized by the presence of petechiæ on the mucous membrane, and by elevations of the cutaneous tissue.

Pathology and Etiology.—Purpura usually occurs as a sequel to debilitating diseases, viz., influenza, catarrhal fever, and strangles; its origin being usually traceable to bad ventilation.

It may also, though rarely, occur as a primary disease, as a result of malhygienic conditions, *e.g.*, defective ventilation, bad drainage, and poisonous effluvia.

Purpura is due to an abnormal condition of the blood and blood-vessels, resulting from the above deleterious influences.

Symptoms.—Purpura may follow the defervescence of mild as well as of severe attacks of such diseases as strangles, catarrh, and influenza. Usually the first noticeable symptom is the sudden development of local swellings on different parts of the body; on the limbs, abdomen, head, and especially around the nostrils, mouth, and lower part of the face. They sometimes appear in patches; but when

on the limbs, they are often more uniformly diffused. They terminate abruptly, not shading insensibly away into the surrounding tissues; they are tense, invariably elevated above the level of the skin, pit slightly on pressure, and are hot and painful. The swellings are due to transudation of blood and serum into the subcutaneous tissue.

Vesicles, or bullæ, of about the size of a pea, appear upon the lower parts of the limbs, around the hock and fetlock joints, and after a time burst and discharge an amber-coloured serous fluid, which scalds and excoriates the surface of the skin over which it flows.

Cracks and fissures also appear at the flexures of the limbs, and from them issues an unhealthy dark-coloured discharge.

The swellings about the sheath, abdomen, and breast often disappear from one part in the earlier stages of the disease, and reappear again in another region.

In many cases the face, lips, nostrils, and eyelids become very much swollen; the swelling ending abruptly about the forehead.

Death may result from swelling of the head owing to interference with respiration, the swollen nostrils causing great difficulty in breathing. The animal, also, is prevented from feeding, owing to the swelling interfering with the movements of the tongue and jaws.

The skin of the swollen parts often sloughs, and large unhealthy sores discharging fetid matter are formed. The lining membrane of the nose becomes more deeply coloured and studded with petechiæ, which gradually coalesce and become darker in colour. Eventually, the greater portion of the mucous membrane of the septum becomes covered over, and there is a discharge of a sero-sanguineous fluid from the nose.

Similar blood extravasations probably simultaneously

involve other mucous membranes and the skin in various parts of the body. The skin being pigmented, the purplish petechial spots are not often seen except in parts where it is not coloured.

The pulse is feeble, often dicrotous and accelerated. There is great debility and prostration. The temperature generally rises 3° or 4° F.

The bowels are at first confined in most instances, but purgation is easily induced. The fæces may be blood-stained, and abdominal pain is frequently present. There is impaired appetite through the course of the disease, and a hoarse hollow cough is often present.

The petechial spots in the nose frequently slough and leave raw surfaces, from which issues the dark-brown sero-sanguineous discharge above mentioned. The tongue is sometimes found swollen, with vesicles on the surface.

The animal moves with difficulty, owing to the stiff and painful state of the joints.

In many cases enteric complications are very severe, and in every case sudden extravasations into the mucous membrane of the alimentary tract may cause death rapidly from excess of the internal hæmorrhage. Similarly, extravasations may occur in connection with other internal structures, and, if extensive, may cause sudden and fatal collapse.

Prognosis.—Is difficult, owing to the fact that cases in which the early symptoms would lead us to expect a favourable issue may at length terminate fatally.

Morbid Anatomy.—The blood is non-coagulable and of dark violet tint; the red cells are altered, and the white cells are increased in number.

The cutaneous swellings and subcutaneous tissues of the swollen parts are infiltrated with dark red, feebly coagulable exudation.

Dark spots are found on the thecæ of the muscles, and in the muscular tissue in various parts.

The serous membranes show petechial spots and extravasations. The pleura, endocardium, pericardium, and cerebral and spinal meninges, may all be so affected. The serous cavities themselves often contain a gelatinous coloured exudation. The mucous membranes are black or blackish-green. The intestinal membranes frequently present ecchymosis, and much infiltration with exudation. The mesenteric glands are similarly infiltrated; the liver and spleen are congested and friable. The lungs are also usually congested, and the trachea and bronchi filled with dark-coloured frothy extravasation.

Treatment.—Attend to the sanitary conditions firstly and chiefly. Medicinally, chlorate of potassium and salines, followed by iron preparations and doses of turpentine, may be administered.

Astringents are preferred by some; ergot of rye, tannin, tincture of perchloride of iron, and acetate of lead being especially recommended.

Antiseptics also are worthy of trial, and the beneficial action of chlorate of potassium may be due to its antiseptic effect.

If there be much debility, alcohol and vegetable tonics should be given. We have found calcium sulphide, in full doses, of very great value in the treatment of this disease.

Ammonium salts are to be avoided.

The diet should be liberal.

Locally, the sores should be dressed with antiseptic solutions. If the head be much swollen, fomentation with warm water is very useful. The swellings should not be punctured.

If there is much dyspnœa from swelling of the mouth or nostrils, tracheotomy may be necessary.

SCARLATINA—SCARLET FEVER.

Definition.—A febrile disease, generally accompanying or occurring as a sequel to some other general and debilitating disorder, and characterized by the eruption of vesicles on certain parts of the skin, and by petechial spots on the mucous membrane of the nose and mouth, together with sore throat and swollen cervical glands.

Varieties.—Scarlatina Simplex, and Scarlatina Anginosa.

Pathology and Etiology.—Scarlatina in the horse has not very much analogy with the disease of the same name in man.

In the horse it rarely occurs as a primary disease, but nearly always is an accompaniment or sequel of some other debilitating disease, such as influenza or strangles. It usually appears as the fever of the primary affection is declining

Scarlatina, like purpura hæmorrhagica, is traceable to malhygienic conditions and weakened constitution associated with the primary malady. It is probably non-contagious, and is more prevalent in spring-time than at other seasons of the year.

Symptoms of Scarlatina Simplex.—Usually begins towards the end of the first week of the primary malady. It may begin on the third, fourth, fifth, or sixth day, or even later. The febrile symptoms become more severe, and there is great prostration, as well as a slightly swollen condition of the eyelids. The temperature is usually elevated about two degrees, and the rise precedes the appearance of the rash.

The 'rash' or 'eruption' of scarlatina take different forms, of which two at least are distinguishable in the horse.

Firstly, there are developed upon the skin of the face,

neck, body, and extremities, smooth blotches, over which the hair is elevated, though the skin itself is little or not at all elevated.

The second form of rash consists in the eruption in various parts of the body, especially on the inner aspect of the thighs, of small rounded vesicles, which eventually desiccate.

The nasal mucous membrane and the membrane of the mouth are covered with scarlet spots, or petechiæ, of variable size. These spots are especially seen on the nasal septum, and on the inner surface of the lips. From the nose there is a serous discharge which gradually becomes yellowish-brown in colour.

The limbs are generally swollen, and the animal is stiff. In some cases of scarlatina there is no eruption, and minute spots on the Schneiderian membrane are all we have to guide us in the diagnosis.

Soreness of the throat is present in most cases.

Symptoms of Scarlatina Anginosa.—Scarlatina anginosa is a severe form of the disease, in which the throat and upper air-passages are especially involved. The symptoms may at first be those of the simple form, and then gradually increase in severity; or the fever may assume a severe form at the outset. In this form the limbs swell rapidly.

The swellings are hot and painful, and may also appear on the face and head.

The rash and vesicular eruption are more frequently found on the limbs than the body, and appear as in the simple form, in successive crops, or spread by the coalescence of neighbouring patches. The petechial spots of the nose and mouth are larger and darker in colour, and more likely to coalesce than in the simpler form of scarlatina. There is great difficulty in breathing and swallowing, and there is loud and painful cough.

The tissues, glands, and other structures in the neighbourhood of the throat and larynx are swollen, infiltrated, and painful. In some protracted cases the bronchial or submaxillary glands may suppurate.

The parts of the skin where the eruption is present are not distinctly elevated, and in this particular they differ from the localised swellings of purpura hæmorrhagica.

The pulse is rapid, varying from 60 or 70 in mild cases, to 100 or more in severer attacks ; it is weak, of small volume, and frequently dicrotous, especially in the later stages.

The respirations are accelerated, especially when the lungs are congested.

Œdematous swellings may suddenly appear on the inferior parts of the abdomen and chest, but they are not of constant occurrence. There is a discharge of serous fluid from the nose, which often becomes yellowish-brown, and in rare cases sanguineous.

The bowels are usually confined.

The fever, when uncomplicated, generally declines in six to eight days ; not suddenly, but by oscillations.

During the rash and infiltration of the structures around the throat the fever shows no abatement.

After the fever, and decline of the rash, there is shedding of the hair where exudation from the vesicles has occurred, and desquamation of the epidermis over the body. Great debility often remains after active symptoms have disappeared, and there is great weakness of the heart, and tendency to effusion into the pleuræ and pericardial sac.

Scarlatina is sometimes followed by inflammation of a rheumatic character.

Prognosis.—Is very uncertain. If the swelling in the throat increases, and the breathing becomes difficult from implication of the upper air-passages, or congestion of the lungs, the prognosis is very grave.

Morbid Anatomy.—The blood is darker, and is not so coagulable as in the healthy condition. The membranes most marked by petechial extravasations are those of the nose and mouth, and those of the heart and abdomen.

The connective tissue in many parts of the body has a reddish or rusty tinge.

The mucous and submucous tissue of the throat, as well as the glands in the neighbourhood, are swollen and infiltrated with exudation.

Diagnosis.—Scarlatina may be confounded with purpura hœnarhagica, erysipelas, and eczema.

The petechiæ in scarlatina are minute spots which may form patches by coalescing together, and they are scarlet in colour. In purpura they are of a dark purple hue. Sore throat is never absent in scarlatina, and but rarely present (unless it be a symptom of previous disease) in purpura.

'Also in scarlatina the swellings are at first in the form of lumps, whereas in purpura they show an even surface, occupying the face as a whole, or the limbs, and terminate abruptly above, as if a cord had been tightly drawn around the part' (Williams).

In scarlatina there is often swelling, and sometimes suppuration of the glands (submaxillary and bronchial glands especially); whereas in purpura this does not occur. Again, the disposition to sloughing of various parts of the body is characteristic of purpura.

Treatment.—Place the animal in a warm well-ventilated loose box. The food should be laxative and nutritious, and doses of nitrate or chlorate of potassium, or sulphite of sodium, may be administered in the drinking-water. This treatment will suffice for mild cases.

In the more severe forms, the throat should be fomented with warm water, and hot poultices should be applied and

renewed frequently. Hot-water vapour, medicated by the addition of carbolic acid, should be used for inhalation.

When the breathing is very difficult, and accompanied by a loud roaring noise, tracheotomy is in some cases necessary in order to avert suffocation. Liquor ammoniæ acetatis, nitric ether, and camphor, may be administered three times daily, in the form of a drench, and a gentle laxative may be given if necessary. If the throat be much swollen and deglutition be very difficult, the remedies mentioned should be given in form of enemas.

As the pyrexial symptoms abate, quinine and other vegetable tonics and sulphate of iron may advantageously be given twice or three times daily.

When suppuration is detected, the abscess should be opened. The external swellings on the limbs should not be interfered with.

During convalescence, careful attention should be paid to the dieting. The food should be of the most nutritious kind, but in moderate quantity at first, until the digestive organs regain vigour. Moderate exercise should be enjoined as the animal regains strength.

RABIES.

Definition.—Rabies is an infective febrile disease, originating in the canine and less frequently in the feline race, and occurring in the horse as the result of a bite of some rabid animal. The specific virus is transmitted through the saliva of the rabid creature; and the disease is characterized by pain in the part bitten, great excitement, irritability, a disposition to bite, clonic spasms, prostration, and death.

Etiology.—Rabies is said by many to arise spontaneously in canine and feline animals, but this is doubtful. It

usually occurs in the horse as the result of the bite of a dog. Whether it can be transmitted from an infected horse to a healthy one by biting is uncertain, but probably it can.

The poison is especially virulent in the saliva. Recently M. Pasteur has demonstrated the existence of a special vegetable parasite in the blood of rabid animals, and to the presence of this germ and its multiplication in the body he attributes this infective disease.

Incubation.—In the horse, the period of incubation varies from two weeks to forty days.

Symptoms.—Begin with great restlessness, excitability, and distress. In some cases there is first observed a lack of power over some of the voluntary muscles, especially those of the hind extremities, or some amount of tonic contraction in the muscles of the back or neck. The excitability is soon much increased, the animal becomes frantic, and attempts in his fury to destroy everything within his reach. In some instances he bites savagely at the seat of the injury.

The temperature is raised 2° or 3°; the pulse is accelerated, its volume is increased, and the artery is firm and hard; the respirations are accelerated, and the appetite is lost; the animal is acutely sensitive to the slightest stimuli. A flash of light or a sudden noise will bring on a paroxysm of fury.

There are convulsive twitchings of the superficial muscles; difficulty in swallowing; a characteristic hoarse cough; abundant flow of saliva from the mouth; paralysis of the posterior extremities may gradually supervene. The remissions and paroxysms in the horse are not so marked a feature as in the dog. In the horse, the remissions are of much shorter duration, and the fits of violence are more aggravated and prolonged than in the latter animal. As

the disease progresses the intervals become still less, until at length the state of fury becomes permanent, and the horse, prostrated, dies in convulsions on the second, third, or fourth day.

Morbid Anatomy.—In most cases there are general congestions and inflammations in connection with nearly every organ and structure of the body. In the abdomen, ecchymoses are found on the serous membranes, the liver and spleen are engorged and abnormally friable, and there are extravasations throughout the alimentary canal; the lungs are congested; the small air-tubes are filled with mucus; the heart shows ecchymoses under the endocardium, and may contain fibrinous clots. There is hyperæmia of the nerve-centres and nerve-trunks, and patchy congestion of the spinal cord and brain, especially at the base and choroid plexus; and there may be effusion into the sub-arachnoid space and ventricles of the cerebrum.

Microscopical examination of the nerve-centres in dogs shows capillary thrombosis and other changes, especially around that portion of the floor of the fourth ventricle occupied by the respiratory centre.

The action of the virus appears at first to involve especially the medulla oblongata and the pneumogastric nerves; thus the difficulty in swallowing and the convulsions of the respiratory muscles are accounted for.

Treatment.—When the disease has set in, treatment is of no avail, and it is advisable to shoot the animal.

Bites by rabid animals should be treated at once. If possible, the tissue around the injury should be excised; if the wound be superficial, the application of caustics will be sufficient; if it be deep, the parts must be first excised, and then cauterized or treated by caustics, such as nitrate of silver, caustic potash, or carbolic acid.

RHEUMATISM.

Varieties.—Acute, Chronic, and Muscular Rheumatism.

ACUTE RHEUMATISM.—Definition.—Is a constitutional fever characterized by the special tendency to inflammation of certain structures, viz.: the articulations, the coverings of the tendons and muscles, the pericardium and endocardium. These inflammations have a tendency to disappear from one part and to reappear in another, and are called metastatic.

Nature of the Disease.—Rheumatism is a general disease, the proximate cause of which has been maintained to be a poisonous substance circulating in the blood. This poison is stated to be lactic, or some other acid. No excess, however, of any such acid has yet been detected in the blood.

Etiology.—*Exciting causes* of rheumatism are: exposure to cold and wet, sudden chills, damp and malhygienic conditions. *Predisposing causes:* a constitutional tendency to rheumatism, or 'rheumatic diathesis,' as it is termed.

Symptoms.—The local symptoms may be preceded by febrile disturbance.

There is sudden and severe lameness, with or without swelling of one or more joints, most commonly the stifle and fetlock, less commonly of the hock and knee. On manipulation the affected joints are found to be excessively tender. The joints may be affected in pairs—for example, the two hock joints or the two stifles may be simultaneously involved, and the lameness is thus symmetrical. The temperature is elevated, and may reach as high as 104°-106° F. The pulse is accelerated, firm, and full; the urine is high-coloured, scanty, acid or neutral in reaction, and loaded with hippurates and hippuric acid; the bowels are constipated.

In most acute cases the heart is affected. If the inflam-

mation be slight, the symptoms of cardiac mischief may pass unobserved. If, however, there be much pericardial effusion, and inflammation of the heart-muscle itself, of the endocardium and valvular structures, very severe symptoms show themselves. There will be great tenderness over the region of the heart, and the sounds will be altered in character (*vide* 'Cardiac Diseases'). The pain and swellings of the joints frequently subside in one extremity quite suddenly, and then reappear in another; this 'metastasis,' or changing of the seat of inflammation, is a marked feature of rheumatism.

Sometimes the inflammation of the joints does not abate and suppuration ensues. In these cases the febrile symptoms become more severe and the joint is destroyed.

Complications.—Inflammation of the pericardium and endocardium ought, perhaps, to be regarded as part of the acute rheumatic inflammation as much as the inflammation of the joints themselves.

Morbid Anatomy.—Death in acute rheumatism is mostly due to cardiac disease, and corresponding lesions are found accordingly. The joints and the structures around them will be found in different conditions of pathological change, varying with the intensity of the inflammation and its duration.

Treatment.—A mild aperient should be given in all cases. A small dose of aloes or a saline purgative may be selected, and the bowels should be kept open by regulation of diet or appropriate remedies. The abstraction of blood is not to be recommended. Bicarbonate of potassium should be given in full doses three or four times daily with the spirit of nitric ether. If the temperature be high, salicylate of sodium given three times daily for a day or two in full doses proves very effectual in reducing it. Colchicum, in the form of a tincture or the wine, and liquor ammoniæ

acetatis are also useful remedies. Locally, hot fomentations to the joints, and anodyne lotions of opium, aconite or belladonna afford much relief. In the later stages, if the joint affections show little improvement, stimulating liniments may be applied. In some cases, where these are ineffectual, vesication with some preparation of cantharides is of service in subduing the inflammation and the consequent pain and tenderness.

CHRONIC RHEUMATISM may follow acute rheumatism, or may occur as an independent affection.

This form may exhibit no pyrexial symptoms whatever. There is less tendency to metastasis of inflammation or to implication of the cardiac structures than in acute rheumatism.

The inflammation of the joints is of a more persistent character, and more frequently leads to ulceration of the cartilages, eburnation of the bones and the formation of osteophytes than is the case in the acute form. In chronic rheumatism the bones in various parts are subject to various pathological changes. Bony excrescences may form on the spinal column, on the bones of the pelvis, and on the synovial membranes of the joints. The joints may, or may not, be enlarged. Anchylosis of the joints, ulceration of the articular cartilages, and eburnation of the extremities of the bones, may often be met with, and various distortions in different parts may result.

Treatment.—When the disease is chronic from the first, occasional laxatives, bicarbonate of potassium and iodide of potassium, may be administered; tonics also may be given in addition. Locally, stimulating liniments are useful. When the disease occurs as a sequel to the acute form, iodide and bicarbonate of potassium, and quinine and other remedies may be tried. Locally it is sometimes advisable to apply the actual cautery.

MUSCULAR RHEUMATISM.—The muscles and their thecæ are liable to a painful affection, which is due to the same causes as other forms of rheumatic inflammation, wet and cold, damp, fatigue. The muscles most commonly involved in the horse are the lumbar and gluteal muscles, and also those of the neck, chest, and shoulder. Generally there is but little constitutional disturbance, though febrile symptoms may be manifested. Especially is this the case when the large lumbar and gluteal muscles are involved, when the pulse is accelerated. The temperature is elevated, and the animal moves with difficulty. The back is elevated, and the affected muscles are painful and tender.

Treatment.—Internally, the same drugs may be prescribed as in acute articular rheumatism, and, in addition, potassium bromide is a valuable remedy. In the chronic forms tonics are very beneficial. Locally, anodyne and stimulating liniments of camphor, belladonna, opium, aconite, with ammonia and turpentine afford much relief.

SCROFULA AND TUBERCULOSIS.

Tuberculosis.—Is an infective disease, characterized by the formation of small nodular masses, called 'tubercles,' and is due to the presence and multiplication of the Bacillus Tuberculosis in the blood and tissues. The lesions are more or less generally distributed, as in acute general tuberculosis, or they are limited to small areas, as in local tuberculosis. Tubercles are of two kinds, grey and yellow.

Grey, or 'miliary' tubercles, or 'grey granulations,' are greyish semi-translucent rounded bodies, varying from minute points to the size of a pin-head, or larger. Yellow tubercles are later stages of the grey variety. They are generally larger, and may reach the size of a chestnut, or even larger, and they are softer.

Fatty degeneration, commencing in the central portions, is the main cause of the difference between the two.

Scrofula.—Is a constitutional condition, characterized by a great susceptibility of certain tissues to become the seat of chronic inflammation, the products of which have but little tendency to become absorbed, but rather tend to infiltrate and accumulate in the tissues.

They thus interfere with the circulation, and so lead to retrogressive and caseous changes; and as there is but little tendency to the formation of fresh blood-vessels, there is no organization of the new growth. These changes are in a great degree to be attributed to the low condition of vitality of the tissues, and to the peculiar character of the inflammatory products.

Relation of Scrofula to Tubercle.—Owing to the similar microscopical appearances of scrofulous and tuberculous lesions, as well as to the presence of the Bacillus Tuberculosis, in what appear to be simply scrofulous products, these two affections have been regarded as identical, and scrofula has been looked upon as not merely due to inherited or acquired liability to simple chronic inflammations.

Now, although tubercular changes are especially liable to occur in scrofulous patients, and though the bacilli may be found in scrofulous lesions, yet the latter may be regarded merely as simple chronic inflammations, affording a suitable soil for the growth of the germ.

Scrofula and Tuberculosis, though of frequent occurrence in cattle, are not commonly met with among horses. In the localized form, tubercle in the horse is said to occur in the lungs, pleura, and glands of the abdominal cavity; and many cases of well-pronounced arthritic disease in young horses are regarded as decidedly scrofulous in nature by Professor Robertson. This form of arthritic disease is

peculiarly an affection of the higher-bred horses, and is due to inherited taint, frequently associated with malhygienic conditions and improper management. It is attended with mal-assimilation of food; and in its treatment special care must be taken to correct the hygienic and dietetic errors. If one of the affected joints be examined, the synovial membrane will be found to be thickened and changed, the joint itself is distended with fluid, and the cartilages and ends of the bones will be found in a condition of retrogressive change.

LYMPHANGITIS.

Synonyms.—'Weed;' inflammatory œdema.

Definition.—A constitutional affection, attended with inflammation, commencing in the lymphatic glands and spreading to the absorbents and blood-vessels of one of the limbs, in most cases a hind one, which becomes swollen and œdematous. In some cases both hind limbs are affected, and, in rare instances, a fore-limb is the seat of the disease. The commonest seat, however, is the left hind-leg.

Nature of the Disease.—'Weed' is to be regarded as a general disturbance of function, especially associated with mal-assimilation and impaired digestion. It is especially a disease of the heavy draught-horses of sluggish lymphatic temperament, and is particularly common among certain kinds of agricultural horses.

An attack renders the animal more subject to a recurrence of the disorder; and in many cases one seizure succeeds another periodically, until the limb assumes a permanently enlarged condition, termed 'elephantiasis.'

Etiology.—Predisposing cause: Lymphatic or sluggish temperament, generally inherited.

Exciting Causes: Foremost among the exciting causes is feeding beyond the requirements of healthy nutrition. Ces-

sation or diminution of work suddenly in well-fed animals is also a common cause, as is seen in frequent occurrence of this disorder among heavy draught-horses after a Sunday's rest. The disease has in consequence been named the 'Monday-morning disease.'

Sudden or prolonged exposure of horses to cold or damp will in many cases bring on an attack by suddenly disturbing the digestive and assimilative functions; and indeed any sudden change in the work or habits of the animal may bring on an attack.

Lymphangitis may also occur as the result of a *local* inflammation of the lymphatics of the limbs, or other parts of the body, as the result of an injury. In the limbs, an injury to the foot, in shoeing, for example, may result in an attack of the lower portion of the limb. This local form does not commence above in the brachial or inguinal glands, as in the general disorder, but spreads upwards along the lymphatic vessels.

Symptoms.—The local inflammation of the limbs is frequently preceded by a rigor, which may last during some hours; and the severity of the disease is shown as a rule by the intensity and duration of this shivering fit.

There is restlessness and lameness at an early stage; and after the rigor has passed, the hot stage follows. The fever is of the sthenic type, and passes quickly through its various stages. The pulse is raised to 70, 90, or 100 beats per minute, and it is hard, full, and firm. The respirations, though not increased in mild cases, are much accelerated in severe attacks, and sweats bedew the body. The visible mucous membranes are congested; the bowels are constipated; the urine is dark-coloured and of high specific gravity. The temperature is raised 2·5° to 3·5° F.; the appetite is lost, and there is great thirst and restlessness.

The swelling and tenderness are first noticed in the lymphatics of the inguinal or brachial region, and the swelling is œdematous, firmish, and slightly irregular. It feels hot, is very tender, and gradually extends downwards, firstly on the inner side of the thigh, but gradually encircling the whole of the limb. The pain and lameness increase until the crisis of the fever is reached, and then remain stationary for a day or two. In severe cases a serous exudation occurs over the inner surface of the limb in the course of the vessels, particularly at the flexures of the joints.

The general and local symptoms continue to increase in severity for twenty-four to forty-eight hours, and then, remaining stationary, are followed in a day or two by defervescence of the fever, and gradual absorption of the local extravasations slowly reduces the swelling.

Lymphangitis is very prone to recur, and with each fresh attack there is less chance of complete recovery, for in many cases the connective-tissue of the limb becomes hypertrophied and indurated. This condition becomes permanently established, and the entire bulk of the limb is augmented, the hair is removed in patches, and the skin also becomes hypertrophied and indurated. From the organized fibroid state of the connective-tissue, this condition has been termed 'Elephantiasis Fibroma,' or 'Elephantiasis.'

Sometimes, after the disappearance of the fever of a first or second attack, abscesses may be formed on the inner aspect of the limb. They are generally confined to the subcutaneous connective-tissues.

Prognosis.—The great majority of cases recover completely, but the fibroid condition may be left as a sequel.

Complications.—Are rare; there may be pneumonia or enteric complications.

Morbid Anatomy.—There is general swelling of the whole limb, the œdema being especially marked above. The subcutaneous tissue and fascia are infiltrated with a yellow coloured lymph, and the tissue itself is hypertrophied and indurated. Similarly the connective tissue among the deeper structures is changed. The lymphatic glands in the affected region are swollen, infiltrated, and hypertrophied. In chronic cases they are firm and indurated; in recent ones they may be soft and broken down, forming small abscesses.

Occasionally the mesenteric glands are enlarged and infiltrated with a gelatinous straw-coloured exudation, and the abdominal cavity may contain a similarly coloured fluid.

In elephantiasis the skin is thickened, and there is organization of the effused matter. The lymphatic glands in these old standing cases may be calcareous.

Treatment.—Bleeding from the jugular vein is generally practised in the early stages, though this treatment is not recommended by Professor Williams. Purgatives should be administered in moderate doses, but excess of purgation is to be avoided. Aloes in doses of two to four drachms may be given, or salines may be used.

Febrifuges are of great benefit in the early stages. A draught of liquor of acetate of ammonia, bicarbonate of potash, nitric ether, and 5 minims of Fleming's tincture of aconite may be administered every four hours. After the purgative has been given, diuretics, to which half a drachm of calomel may be added in cases which have not followed some debilitating disease, may be administered once daily in addition. Tincture of colchicum is recommended by some. The affected limb should be fomented with warm water, and if the pain is severe, tincture of opium may be added to the water, or it may be applied with other anodynes as a lotion after each fomentation.

The diet should be carefully attended to. In the early stages a restricted and cooling diet should be ordered, but in the later stages, when debility supervenes, the diet should be nutritious and well regulated, and the limb should be supported by bandages applied pretty firmly.

Where the limb is much thickened, iodide of potassium or tincture of iodine may be given internally, and the ointment of iodine may be used externally, and continued while the animal is at work.

BURSATEE.

Definition.—Bursatee is a disease probably due to a vegetable parasite belonging to the moulds or hyphomycetes, characterized by peculiar slow structural changes in external wounds, in the subcutaneous tissue, or in internal organs, or in all these situations.

Distribution.—Bursatee is an Indian disease probably peculiar to the horse, deriving its name from 'Bursat,' which signifies 'rain,' from the association of this malady with the rainy season. But probably it is most frequent in the time just preceding the wet season. It occurs throughout the Indian Empire, especially, however, in the northern parts, and is more commonly met with on the plains than in elevated situations.

Etiology and Nature.—Bursatee is in all probability due to a special fungoid growth, and in the *Veterinary Journal* of July, 1884, Mr. F. Smith says that he has found a mould fungus in every fresh specimen of the sore which he has examined. The filaments of the mould, or hyphæ, as they are termed, are here recorded to be $\frac{1}{20000}$ inch in diameter, and of great length. He has not observed any spores. In the propagation of bursatee, flies have been asserted to play a prominent part, and water also is said to be a means of disseminating the disease. The lesions found in bursatee

have been compared with cancerous growths, but there appears to be but little analogy between them. Malhygienic conditions appear to favour the development of the disease, and it is said to be commoner in stabled animals. Whether bursatee can be transmitted from parent to offspring is an unsettled question. Bursatee is probably non-contagious.

Symptoms.—The special feature of bursatee is the appearance of peculiar growths termed 'kunkurs.'* These are mainly found in those situations which, from their position, are most liable to be the seat of wounds from any cause. The subcutaneous tumours are first noticed as soft nodulated swellings. In about eight or ten days from their first appearance they become hardened, until they become as firm as fibroid tumours. After a variable period the tumour ulcerates, and a bursatee sore or ulcer results. The sore is indolent, and has a papillated appearance, and discharges a small quantity of pus. The edges of the ulcer are slightly raised and undermined, and on the floor are scattered little hardened eminences called kunkurs, which may be squeezed out. The sores have but little tendency to spread, though in some cases they do increase in size, and neighbouring sores thus become confluent.

The reparative material is also of the nature of the kunkurous growth, and the cicatrix formed in healing is of a greyish colour.

Ulceration and cicatrization may go on together in different parts of the sore, and the whole scab may be thrown off, forming an unhealthy ulcerated surface, or it may be fissured, and discharge a grumous matter. The inner canthus of the eye is often the seat of the affection, and from thence it may cover a considerable area of the face;

* *Kunkur* is the native term for a stone used in India for economic purposes.

probably the irritation of the tears sets up a condition suitable to the growth of the fungus. The angles of the mouth are also frequently affected, and the bit may cause the original injury, which afterwards takes on a bursatee form. The legs, from the knee downwards, especially the fetlocks and coronets, are often affected, and in these situations the growth has a tendency to assume more the character of bone than in other parts. The vagina, urethra, glans penis and prepuce are also frequently affected. The site of an old sore always affords a soil for the peculiar growth in affected animals, and fresh wounds similarly undergo the above changes. Cicatrization rarely or never occurs spontaneously, and with the return of the season there is great probability of recurrence of the disease on some other part of the body. A cicatrice, if external, may lead to impairment of function of the parts involved. The general condition varies in bursatee, but there is usually more or less debility.

Morbid Anatomy.—The lesions of bursatee are found in external wounds, in subcutaneous tissues, and in internal organs, or in two or all three of these situations. They may be found in the internal organs of animals which have shown no signs of the disease during life. In size, the growths vary from a millet seed to a filbert nut. In the early stages, microscopical examination shows them to be composed of two kinds of cells. One kind of cells have a tendency to elongate into fibres, the other have abundant nuclei and nucleoli. In the next stage the growths are firmer; the section is yellowish white. Many of the more stable and less active cells have become converted into fibrils, while the other cells, dispersed among them, are of a brownish colour.

This is the stage of soft kunkur. In a still later stage, the tissue is harder, and the greater part of it is in a con-

dition of calcareous metamorphosis. In the internal organs, the kunkurous growth has been observed to have a fibrous capsule. The growths have been found in the liver, spleen, mesenteric glands, cæcum, lungs, pleura and bronchial glands, salivary glands, and submucous tissues of the urethra, and in other situations.

Treatment.—Attention to the hygienic conditions and nutritious diet are of first importance. Tonics may be given from the onset of the disease. The sores should be dressed with antiseptic lotions of carbolic or sulphurous acid, or eucalyptus, etc. All vegetating growths and masses of kunkur should be excised or removed by the actual cautery. It is essential that the whole of the growths should be excised, as otherwise the growth will recur. After excision the wound may be dressed with lunar caustic or sulphate of copper in order to stimulate the healing process.

DIABETES.

Varieties.—(1) Diabetes insipidus, or polyuria or hydruria.

(2) Diabetes mellitus.

Definition.—Diabetes is a diseased condition accompanied by excessive secretion of urine, great thirst, emaciation, and debility, and in the second variety characterized by the presence of grape sugar or glucose in greater or less amount in the urine.

DIABETES INSIPIDUS—Nature of Disease.—Diabetes insipidus is the commoner of the two forms of diabetes in the horse, and is regarded by some merely as a functional disease of the kidneys. It is, however, rather to be looked upon as a general affection due to some disordered condition of the assimilative processes.

This disease is so common among horses under certain

conditions, that although usually a sporadic affection, it occasionally develops truly enzootic characters (Robertson).

Etiology.—It is probably caused by disordered condition of the vaso-motor centres, leading to dilation of the renal vessels, and thus causing polyuria. The disordered innervation is probably due to defective assimilation, brought about by improper diet or by causes peculiar to the locality.

Mouldy hay, musty, damp, or kiln-dried oats, and bad corn, are fertile sources of the disease. Boiled food is also sometimes a cause of diabetes insipidus. In some cases the disease may come on in the absence of such causes, and it is then of a most severe type, and usually due to mal-assimilation of food and other causes, such as prolonged exertion and exposure to cold.

During convalescence from some debilitating diseases, even slight dietetic errors may induce polyuria.

Symptoms.—Excessive urination, great thirst, anorexia or depraved appetite, are the diagnostic symptoms. The mouth has a sour, unpleasant odour, and the skin is scurfy. The pulse varies in number, but the volume is diminished and the tone deficient. The visible mucous membranes are pale, and the animal is enfeebled and perspires on slight exertion. The urine voided is pale, of low specific gravity, from 1002 to 1003, but the total amount of solids is probably not diminished. According to Lassaigne, it contains free acetic acid, and alkaline carbonates are absent. When this form of diabetes follows some debilitating disease, the urine is of higher specific gravity, and the debility and wasting are more marked.

When the disease continues unchecked, the animal gradually loses strength, becomes emaciated, and structural changes follow in many organs. Dropsical effusions into the serous cavities and the lax connective-tissues are then of common occurrence.

Morbid Anatomy.—General pallor and loss of tonicity of the muscles, and soft flabby condition of the large glands of the abdomen, are noticed. In the brain and spinal cord there may be serous effusion into the sub-arachnoid spaces, or infiltration of the membranes with a gelatinous matter.

Treatment.—Change and careful regulation of the diet are essential. At first the food should be restricted to hay and mashes, and the animal should be rested and carefully attended to for a few days.

Mild aperients should be given, and bicarbonate of soda or potash allowed freely in the drinking-water. The alkaline salt is especially called for when the horse exhibits a depraved appetite by eating the mortar or licking the walls.

Iodine and iodide of potassium are the most valuable of internal remedies, and, indeed, are regarded as specifics. The compound tincture of iodine may be prescribed, or iodide of potassium may be given in the drinking-water in two-drachm doses twice daily. Vegetable tonics may also be administered.

Other remedies often used are sulphate and perchloride of iron, and arsenic in the form of Fowler's solution.

DIABETES MELLITUS.—This disease is rarely met with in the horse, and its pathology is very obscure.

Etiology.—The glycogenic activity of the liver appears to be increased in this disease, and thus more glycogen is converted into glucose and thrown into the circulation than can be used in the economy. The surplus is thus excreted by the kidneys. Whether this increased functional activity is due to direct nervous influence on the hepatic cells, or whether it arises from vaso-motor disturbance producing dilation of the hepatic arteries, or lastly, whether it be in some cases due to the former, and in other cases to the latter, is uncertain.

Symptoms.—There is excessive elimination of urine, indigestion, thirst, and the various other symptoms of polyuria are present. The urine has a fragrant smell, and is of higher specific gravity, and when tested is found to contain glucose or grape-sugar. It may be tested for by Trommer's test, or by using Pavy's test pellets, now specially manufactured for the purpose. The fermentation test may also be applied.*

Treatment.—The diet should be nourishing, and as nearly as possible devoid of purely saccharine or starchy matter. Opium or codeia, one of its alkaloids, may be given twice daily. Mineral acids, iron salts, and astringents, as gallic acid, are also recommended.

AZOTURIA.

Definition.—Is a diseased condition characterized by tonic or clonic spasms of the large muscles of the posterior part of the body and of the limbs, and by the elimination of nitrogenous, darkly coloured urine of high specific gravity.

Nature of the Disease.—Azoturia is the result of an oversupply or excess of proteid material in the system.

The muscular elements are probably more involved than the other structures of the body, and the condition of the urine is the result of the pathological changes which have taken place.

The phenomena of the disease are intimately associated with mal-nutrition, and may originate from this cause, or also from a disordered condition of the nerve-centres, or from both causes.

Etiology.—The primary cause of azoturia is dietetic. The disease may attack animals out at grass, and it is especially apt to follow periods of idleness preceded by active work. It does not so much occur during the actual rest as when

* For particulars as to examination of the urine, see 'Urine in Health and Disease.'

the animal again resumes work. Tares, vetches, and other leguminous vegetables, are especially apt to induce this serious malady. Horses fed with materials rich in proteids during a state of rest are the most subject to attack. It is most common during autumn, and it has been observed to be of more frequent occurrence among mares than among geldings.

This is especially the case during the œstrum, when the excitability of mares is increased.

Symptoms.—The symptoms of azoturia come on suddenly, without any previous warning or indication of disease. Horses may be attacked when in the stable, or, as more commonly occurs, when they have travelled a short distance after a period of rest.

In the latter case they become suddenly lame, or have such difficulty in moving the limbs, generally the hind ones, that they are scarcely able to move. In some cases they are, as it were, struck down suddenly from loss of motor power in the posterior extremities. In other instances the attack is not so sudden; the animal becomes very restless, perspires freely, and shows a disposition to lie down, and very shortly the characteristic symptoms appear, if not already present. There are tremors and violent spasmodic twitchings of the large muscles of the loins and gluteal muscles, ending in tonic contraction or more or less total loss of power. The perspiration may be excessive. The urine discharged is high-coloured, thick, and of a brownish tint. The specific gravity is always much increased, and may reach 1185 or even more, and is highest during the first few hours after the attack. It often gradually diminishes afterwards.

The pulse varies from 60 to 80 beats per minute, and is generally weak, though in some cases, on the contrary, it is strong.

The temperature varies from 102° to 104-5° or even higher.

The bowels may be regular, and in slight attacks the appetite is not impaired. In severe cases the animal lies prostrate, generally unable to rise; refuses to eat or drink; struggles violently in his attempts to raise himself, and partial coma may then supervene. There is also in such instances much congestion of the conjunctivæ. While the animal remains thus prostrate, there are periodical strainings and ejections of small quantities of urine, or there may be constant involuntary dribblings.

The urine, if carefully examined as soon as possible after being voided, will be found to be loaded with urea. Indeed, so abundant is this product, that if nitric acid be added to a portion of urine in a vessel, the whole becomes almost solid, from the formation of crystals of nitrate of urea. Albumen is not generally found, though in some cases the solidification is in part due to the coagulation of a small amount present.

The crystals of urea-nitrate appear as mica-like rhomboid scales of a brownish colour, and fall to the bottom of the vessel. At first the addition of the acid causes effervescence, from the presence of ammonium carbonate in the alkaline urine, but after the effervescence is over, urea is readily precipitated.

If the serum of the blood be examined, crystals of nitrate of urea may be formed by extracting the urea from the serum and adding a few drops of nitric acid.

Prognosis.—Is very serious in the sudden cases. In some instances the animal is struck down, struggles violently for a few hours, and then becomes comatose and dies.

In other instances, again, the severe symptoms abate; but the animal does not regain the use of its limbs, and although it may eat well and be perfectly conscious, it is unable to rise, and eventually dies from some complication.

In favourable cases the urine gradually becomes normal, the spasms gradually lessen in severity, the power of movement returns, and in a few days the animal is convalescent.

In other cases for a long time the loss of power in certain muscles remains. Not in all cases is there recovery when the urine becomes normal, for the cerebral complications may still continue, and death ensue in consequence.

Diagnosis.—The only diseases which are likely to be confounded with azoturia are anthrax and cerebro-spinal meningitis.

Morbid Anatomy.—There is general congestion throughout the body, especially in the glands of the abdomen. The bladder contains thick brownish fluid, and its lining membrane is similarly stained. The nerve-centres may be more or less hyperæmic, and the blood is found to be dark and semi-fluid.

Treatment.—The animal should be placed in a well-ventilated loose-box, with plenty of straw.

It is of first importance to act freely on the excretory organs. For this purpose, a cathartic or oleaginous purgative should be given at once. A full dose of aloes in the form of a ball is probably the best aperient in this condition. Enemas should also be administered at intervals until purgation begins.

If there be much fever, the liquor of acetate of ammonium or other febrifuge may be given in the drinking-water, which should always be kept near the animal, and should not be restricted in amount. Stimulants and sedatives are harmful in the early stages, and should not be given; but if on the third or fourth day the animal shows no signs of improvement, moderate doses of alcohol or of nitric ether should be allowed three times daily. In cases where there is any indication of failure of the heart's action, stimulants

should be given freely; but ammonia and its compounds are not to be recommended, as the blood is already surcharged with nitrogenous waste-products.

If the animal is unable to rise, he should be raised by means of slings, in which he may be allowed to remain for a few days. He may be taken out occasionally for a little exercise. If he seems disinclined to stand, the limbs may with advantage be briskly rubbed.

Fomentations on the loins by means of woollen cloths wrung out from warm water are recommended by some, but this treatment is viewed with disfavour by Professor Williams, as likely to increase the irritability of the animal.

The diet for the first few days should be restricted, and of a light, easily digestible kind. When convalescence is being established, it should be moderately nutritious. A few doses of nux vomica may be given with advantage after the discontinuance of the febrifuges.*

OXALURIA.

Definition.—Is a condition of the system characterized by the presence of oxalate of lime in the urine, debility, loss of flesh, stiffness in the loins, and a branny scurf on the surface of the body.

Etiology.—The causes are: irregularity of feeding; ir-

* We believe pilocarpin injected hypodermically to be of great value in the early stages of the severe forms of azoturia, in addition to the cathartic remedies and febrifuges, though as yet we have not had sufficient experience of this disease to assert that this remedy may always do good. It is a powerful diaphoretic, and acts by enabling the blood to throw off the excess of effete matter.

The phenomena of azoturia, indeed, seem to be dependent upon the accumulation of urea in the blood, owing to its increased production, and also to failure of the kidneys to eliminate this excess. Further, they are probably dependent upon the decomposition of this substance in the blood.

regular exercise and work; want of sufficient exercise; improper diet, and impaired digestion from any cause. Oxaluria is most commonly met with in hunters, or other horses whose work is irregular, and which undergo long periods of fasting. It is also induced by food rich in saccharine matter, as carrots, turnips, or other roots, especially if the digestion be deranged.

Symptoms.—The animal is stiff in the loins, and easily fatigued; he loses flesh, the skin becomes dry and scurfy, the appetite is capricious, and there is frequent voiding of a pale-coloured urine, often accompanied by uneasiness.

The bowels are often constipated, and the tongue furred.

The urine is clear, pale, and acid or neutral in reaction, and when examined under the microscope shows crystals of oxalate of lime. These are either in the form of octahedra or very rarely dumb-bell shaped.

Treatment.—Firstly, it is necessary to administer a purgative. The diet should be carefully regulated, and roots and food containing much sugar should not be allowed. Vegetable tonics and mineral acids should be given to improve the digestion. For this purpose dilute nitro-hydrochloric acid, tincture of nux vomica, and gentian may be administered three times daily. If the drinking-water contain lime in excess, it should be changed, as other remedies will not effect a cure if there be an excess of lime in the system.

Regular exercise and fresh air are essential.

STOMATITIS PUSTULOSA CONTAGIOSA.*

Several cases of this contagious disease are reported as having occurred at Berlin during the summer of 1876.

* For full description of this disease, vide *Veterinary Journal* for November, 1878.

The horses were from four to five years old. Some of them stood with depressed and outstretched heads. The ears and extremities were cold. The pulse was about 60 per minute, and the respirations were normal. There was no loss of appetite even in the worst cases, but the animals masticated slowly, swallowed with difficulty, and saliva in abundance was secreted.

There was a greenish discharge from the nostrils, and when water was taken, much of it returned through the nose.

The Schneiderian membrane was of a rose-red colour. The submaxillary glands were enlarged on both sides; they were hard, firm, and about as large as hen's eggs. They were not attached to the skin or bone. The oral mucous membrane was hot, red, and covered with a tenacious secretion, and many small firm nodules, varying in size from a millet to a lentil seed, could be felt in it. The nodules increased in size and number rapidly, and soon appeared on the cheeks, and on the upper and lower surface and frænum of the tongue. In one or two days later the summits of the nodules presented a whitish appearance, or the epithelium was removed and a small ulcerated surface became apparent. In four or five days later the ulcerations were numerous, the dental surface of the lips, and the top of the frænum and inferior surface of the tongue being especially affected. About the sixth to eighth day the ulcerations began to heal. The disease ran its course rapidly, and all the cases under treatment recovered in from twelve to fourteen days.

This disease, on superficial examination, might be mistaken for glanders, but there are important differences between the two. In the former the ulcers were round, with sharply-defined borders, varying in size from a fourpenny-piece to a shilling, and the edges were neither

serrated nor swollen, as in glanders. Further, the ulcers were superficial, rarely reaching as deep as the subcutis, and they readily healed after the sixth day. They were isolated and independent of the course of the lymphatics, and when the brownish scab was removed they presented a whitish granular surface. The disease only lasted from twelve to fourteen days, and ran through a whole stable in three weeks or so.

This disease, therefore, differs from glanders in the character, distribution and rapidity of the healing of the ulcerations; in the absence of inflammation of the absorbents; in the absence of ulcerations on the Schneiderian membrane, and finally in the rapid course of the disease, in the shortness of its latent period, and in the rapidity with which it spreads from one animal to another.

Stomatitis pustulosa also resembles variola equina, but it cannot be regarded as true equine variola.

CHAPTER V.

DISEASES OF THE RESPIRATORY ORGANS.

METHODS OF PHYSICAL EXAMINATION, AND THE SIGNS AFFORDED BY THEM.

THE various methods of physical examination of the respiratory organs are :

INSPECTION.—When we inspect or look at the chest, we have two sets of facts to observe—

Firstly, the shape or form of the chest;

Secondly, the number and the character of the movements of the chest during respiration.

When the respirations are increased in range above the normal, they are said to be exaggerated, as, for example, in certain febrile affections; when decreased below the normal, they are said to be deficient or impaired, as, for instance, in certain diseases of the brain.

When the movements are deficient, less air enters the lungs than is necessary, and dyspnœa, or shortness of breath, will result.

Dyspnœa may result from two opposite conditions; the lungs may either be prevented from expanding or from contracting as much as in the normal condition. The former is termed 'defective inspiration' or 'deficient expansion,' and the dyspnœa is said to be 'inspiratory;' the latter is termed 'deficient expiration,' and the dyspnœa is called

'expiratory.' Again, disturbance of the relation between the thoracic and abdominal movements may be detected. Thoracic movements in excess may be due to interference with the action of the diaphragm or abdominal muscles, as in peritonitis. Abdominal movements in excess may result from causes impeding the action of the chest-walls, as in pleurisy.

PALPATION.—By palpation, or the application of the hand to the walls of the chest, we may feel the shape and movements of the walls, and whether there be any tenderness or not. Occasionally also abnormal sensations, as pleuritic friction, may be felt.

MENSURATION.—This method is not much practised in veterinary medicine in this country, but is recommended by French veterinarians.

PERCUSSION.—This method consists in tapping the walls of the body so as to cause them to yield a sound. Percussion may be either direct (immediate), when we strike the surface with the tips of the fingers, or with the knuckles of the closed hand; or it may be indirect (mediate), when we percuss upon something placed upon the skin.

In the direct method, which is the more convenient in examining horses, it is best to strike over the surface of a rib, and as perpendicularly as possible. In the indirect method, the intervening body, whether it be a finger or a plesameter, should be pressed closely to the surface of the body.

If the walls of a cavity containing air be struck, a hollow, resonant, or low-pitched sound is obtained. On the other hand, if a solid mass, as for instance the thigh, be struck, the sound produced is dull, non-resonant, or high-pitched. A very resonant note is said to be tympanitic (*tympanum*, a drum).

If the walls of the chest be percussed where the lung is

in close contact with the walls, a fairly resonant note is heard in health, the sound depending on the vibration of the air in the pulmonary tissue, and on the vibration of the chest-walls themselves.

General increase of resonance occurs in the chest in cases of pulmonary emphysema. Partial increase may occur over areas where the lung has become more functionally active, and dilated in consequence, from loss or impaired function of the rest. Diminished resonance may occur from any cause obstructing the entrance of air into the vesicles of the lung. Loss of resonance may be due to hepatization of the lung or to effusion into the pleural cavity.

AUSCULTATION.—By auscultation we mean listening to the sounds of the interior, either directly, by application of the ear to the surface, or indirectly, by means of a stethoscope. In the normal condition, auscultation of the respiratory organs of the horse reveals at least three typical sounds :

1. Tracheal, Laryngeal or Nasal Breathing.

This may be heard over the larger cavities of the head, the larynx, and the trachea.

It is a soft blowing murmur, audible during inspiration and expiration, between which there is a short interval.

2. Bronchial or Tubal Breathing.—This breathing is harsher, and of shorter duration and less intensity, than the tracheal. The interval, also, between inspiration and expiration, is less marked. It is best heard at the bifurcation of the trachea, at the anterior part of the chest.

Over the superior and middle third of the chest it is less characteristic, and is most distinct in the upper third.

3. Pulmonary or Vesicular Breathing, or Respiratory Murmur.—The respiratory murmur is due to the entrance of air into the vesicles, or to conduction of the laryngeal

sounds along the bronchi and small tubes, and their modification by having to pass through the vesicles, or it may be due to both these causes.

It is a characteristic soft breezy murmur, heard in inspiration and expiration, between which there is an appreciable interval. The expiration is very faint or inaudible. The murmur is louder in the young than in aged animals, and may be heard in the horse over the greater part of the chest, and is most distinct in well-bred horses, and in those in which the chest-walls are not loaded with muscle and fat.

Changes in the Breathing Sounds in Disease:

1. In the Anterior Air-Passages.—In some horses the nasal sound is abnormally increased in exercise. This sound is louder in expiration than in inspiration. It is best heard during trotting or cantering gently, and usually disappears when the horse is going at a good steady gallop.

It arises from the flapping of the nostrils, and constitutes what is improperly termed 'high-blowing.' Professor Williams says, 'I have repeatedly observed that some horses will emit the sound when first put into work after resting a while, but when brought into condition the sound entirely ceases, and I have thought that the sound was due to flaccid condition of the dilatores naris muscles.'

High-blowing may also be due to narrowness of the nasal apertures and passages.

These conditions as they do not interfere with the usefulness of the horse, cannot be said to constitute unsoundness.

In thickening of the Schneiderian membrane, or when abnormal growths occupy the air-cavities of the head, a more or less blowing sound may be heard.

In some instances, if the obstruction be very great, the sound may become of a whistling character; this, however, is rare. It may be mistaken for a sound originating in the

larynx or lungs, but auscultation will tell us where the sound is produced.

Snoring is diagnostic of polypus, and snuffling sounds indicate some disorder, accompanied by discharge from the nose, such as catarrh, purpura hæmorrhagica or glanders, scarlatina, etc.

A dry wheezing or whistling sound, which persists, indicates narrowing of the aperture of the larynx.

In some cases of asthma a wheezing vibratory noise may be heard along the trachea.

2. In the Bronchi and Lungs.—In ausculting the chest, the normal respiratory murmur may be heard to be augmented, and this augmentation may be local or it may be general.

A general increase may be heard as the result of anything which for a time enhances the activity of the breathing. When it is increased over the surface, as the result of the exclusion of air from a neighbouring portion of the lung, it is termed supplementary. When heard over parts of the chest, where it is not audible in health, the murmur indicates an infiltration or solidification of the pulmonary tissues.

The respiratory murmur may also be diminished, and this diminution may likewise be partial or general.

General diminution may be due to various causes, for example, to lessened respiratory action, such as occurs in many cerebral disorders; or it may be due to obstructions, by which the normal expansion of the chest is lessened. The murmur may be entirely suppressed from a variety of causes. The suppression is one of the signs of consolidation of the pulmonary tissues and of pleural effusion.

Again, the murmur may lose its regularity and rhythmic character. It may become oscillating or interrupted in various ways in certain phases of pleurisy, or the expiration

may become prolonged, as in emphysema. Finally, the murmur may be altered in character.

If it become of higher pitch, louder and more prolonged in expiration, it is then said to be '*exaggerated*' or '*harsh breathing*,' and indicates changes in the consistence of the lung-tissue, depending upon compression in the early stage of bronchial and pulmonary inflammation. This harsh breathing may subside and the breathing become normal; or, on the other hand, if the disease progresses, it may become replaced by a very characteristic kind of breathing, very important in diagnosis, termed '*Bronchial breathing*.'

Bronchial Breathing is similar to the variety of breathing above described as being audible in the normal condition at the bifurcation of the trachea at the anterior part of the chest, but is louder and more distinct.

When it is heard over parts of the chest where it is normally absent, it indicates disease, and is a marked sign of consolidation of the lung. Sometimes it is heard in emphysema.

Cavernous and amphoric breathing are varieties of breathing only heard in disease. They approach the normal laryngeal or nasal sound, but they have a greater hollowness. They are only heard in rare instances, in cases where the air-tubes are in connection with cavities in the lung-tissue.

New Sounds heard in Auscultating the Respiratory Organs.—We have as yet only spoken of sounds the analogues of which may be heard in health. We have now to consider sounds only heard in disease. They fall under two headings: (1) Those produced in the lungs, pulmonary; and (2) those produced in the pleural cavity.

1. The Pulmonary Sounds.—To these sounds the term 'râles' is applied; and they may be divided into crepitation, rhonchus, and sibilus.

Crepitation.—Crepitation is produced by the bursting of air-bubbles in fluid, and is spoken of as large, medium, and small, according to the supposed site of the bubbles which produce them. Crepitation has, therefore, been spoken of as moist sounds in contradistinction to rhonchus and sibilus, which are dry sounds.

Very fine crepitation is, however, probably due to the separation of the walls of the air-vesicles in inspiration, when, as the result of pressure or collapse, they have been in partial contact. Crepitation similar to this, but not similarly produced, is also occasionally heard over emphysematous lungs.

Rhonchus and Sibilus.—These sounds are due to vibration in the air, through bronchial tubes in which the mucous membrane has become altered or roughened by inflammation or secretion.

Rhonchus is produced in the large tubes and is deeper in tone. It is described as a snoring, cooing, or grating, according to its special character in different instances.

Sibilus is produced in the smaller tubes, and is a wheezing noise, more hissing in character than rhonchus.

2. Pleural Friction.—In health the surfaces of the pleuræ are smooth, and move upon each other without producing any sound. When the pleuræ are roughened by disease, the rubbing of the two roughened surfaces produces a sound to which the name of 'friction' is applied. Friction, being produced in the pleura, sounds close under the ear, and is double, owing to the movement of the lungs to and fro.

It may be very limited in extent.

When effusion into the pleural cavity takes place, the surfaces of the serous membranes are separated, and the friction thus disappears; but when the inflammation sub-

sides, and the fluid is reabsorbed, the two layers come into contact, and the friction is heard again, and is termed 'redux friction.'

Other Sounds sometimes heard in Auscultating the Chest under Certain Conditions. — Metallic Tinkling is a sound caused by drops of fluid falling from the walls of a cavity into the fluid below. It resembles the sound produced by drops of water falling into a well.

Gurgling Sounds are due to the liberation of air-bubbles from effused material.

Cough Resonance.—If we listen to the walls of the chest of a healthy horse, as he coughs the sound is heard as being of a soft, dull, indistinct character.

In pulmonary disease the character of this cough resonance is altered. For instance, in hepatisation, or compression of the lung from effusion, if the horse be made to cough while we listen to the chest, the sound produced is much louder and more distinct and harder over the involved area than in the normal condition.

CERTAIN SYMPTOMS ASSOCIATED WITH DISORDER OF THE RESPIRATORY ORGANS.

GRUNTING.—If a horse when struck or suddenly moved makes, during expiration, a grunting noise, he is said to be a 'grunter.' The emission of this sound is always to be looked upon with suspicion, as it generally accompanies whistling and roaring. It may or may not be due to disease of the larynx. A horse may grunt from pain, when suffering from pleurisy, pleurodynia, and other disorders, and some horses grunt when struck at or moved suddenly.

A large number of cart-horses, and large horses of all breeds, are apt to be grunters being nevertheless quite sound in their wind. Any horse also may grunt if he has been fed for a time with bulky food.

COUGHING.—Cough is a modification of breathing, characterized by a deep drawn inspiration, followed by closure of the glottis, and one or more short but violent expiratory acts. It is generally excited by irritation or abnormal accumulation at the glottis, in the trachea, or in the larger bronchial tubes, or it is a simple nervous affection.

Cough is dry or moist. Dry cough is of several varieties; short, hollow, hacking, broken-winded, and spasmodic. Dry cough is characteristic of irritation, and of dryness of the respiratory mucous membrane. In the early stages of laryngitis it is long and loud and sonorous, becoming rasping, and afterwards moist.

In chronic diseases of the larynx it is loud and often hollow. In the early stages of bronchitis it has a hollow metallic sound, and becomes moist afterwards, and is more or less painful throughout the disease.

In pneumonia the cough is short, and in the later stages of the disease it is accompanied by expectoration of a rusty-coloured tenacious secretion.

The cough of pleurisy is dry and hacking, and is sometimes broken, as it were, in the middle. The broken-winded cough is at first spasmodic, becoming, as the disease advances, feeble, short, and single: the horse, being unable to relieve himself by the action of the chest and lungs, gives a suppressed cough, which is characteristic. The hollow cough varies in intensity, and is indicative of chronic disease. Moist cough is indicative of an inflamed and humid condition of the respiratory mucous membrane.

WHISTLING is of two kinds—soft or moist, dry or

hard. Soft whistling occurs in acute laryngitis, when there is considerable increase of mucus, and also when the mucous membrane is temporarily swollen. If it be occasioned by a large amount of mucous secretion, it is of a wheezing character, and is mostly diminished when the horse coughs. If it arises from inflammatory infiltration of the laryngeal mucous membrane, it is louder during inspiration than in expiration.

Soft Whistling constitutes temporary unsoundness, and it is, in many cases, unsafe to hazard a decided opinion for some days or even weeks, until the thickening of the mucous membrane and relaxation of the vocal cords has had time to subside.

Dry Whistling.—According to some authorities, dry whistling is to be regarded as a modified form of roaring. Others, however, are of opinion that whistling and roaring are due to different pathological conditions of the larynx, and that they may exist independently of each other. With the latter views we entirely concur.

Dry whistling, like roaring, is a noise made more especially during inspiration. It is due to diminished calibre of the larynx, and sometimes of the trachea, owing to permanent thickening of the mucous membrane, distortion of the neck by tight reining, the presence of a tumour in the trachea, or to any cause which diminishes the calibre of the passages through which the air passes to and from the lungs.

Whistling, though loudest in inspiration, is by no means always absent during expiration.

If entirely absent during expiration, the pathological change is to be sought in the larynx, or upper part of the air-passages.

Whistling, like roaring, is often traceable to hereditary taint, and constitutes unsoundness.

ROARING is a loud unnatural sound made during inspiration. It is in some cases symptomatic of acute laryngitis, and then only indicates temporary unsoundness.

Roaring is generally due to atrophy and fatty degeneration of certain muscles of the larynx, especially those which pass between and attach the arytenoid cartilages to the cricoid posteriorly. The origin of the degenerative changes in the muscles leading to the paralysis is not certainly known. The disease is generally confined to the left laryngeal muscles, and many have supposed that this is due to the fact that the recurrent laryngeal nerve passes from the pneumogastric at a more distant spot than does the corresponding nerve of the right side, and that, moreover, the nerve on the left side winds round the posterior aorta. According to this view, the left recurrent nerve is thus more likely to be involved in any disease of the chest than on the right side, where it is given off opposite the first rib, and winds round the dorsal artery.

Such an explanation, however, is open to many objections; firstly, roaring is not a common sequel of pulmonary disease; secondly, many roarers have never had any chest affection; thirdly, mares and ponies are less liable to become roarers than large geldings and stallions, but are equally prone to chest affections; fourthly, the nerve itself shows no pathological change, as far as we know; and finally, the atrophic change in the muscles, though usually seen on the left side of the larynx only, is not uncommon on the right side also, though to a less extent.

The disease of the muscles is, however, of an essentially nervous origin, and ought to be classed as a form of wasting palsy—paralysis atrophicans.

Roaring is in most instances gradually developed. At first the sound may be intermittent, and even weeks may

elapse before it recurs after being once heard. As the muscles continue to waste, there is a corresponding and permanent loss of power, and what at first was intermittent is now established.

Most commonly, however, the noise is not intermittent. It is usually slight at first, and then gradually increases with the waste, and consequent inability of the muscles to open the laryngeal orifice.

Causes of Roaring.—Heredity plays a very prominent part in the transmission of roaring. Professor Williams mentions a breed of horses in which nearly all the animals of both sexes are roarers. Horses and geldings are, however, more likely to become roarers than mares, which are rarely afflicted unless the hereditary predisposition is very marked.

Small ponies are rarely if ever affected. An animal predisposed to roaring in most cases suffers from laryngeal affections from very slight causes, and the infirmity usually appears after several attacks of sore throat and cold.

There are other causes of roaring besides those resulting from the condition of the laryngeal muscles above mentioned.

These are not so frequently met with, but are of great importance.

The characteristic sound may be due to disease of the nose: nasal polypi; osseous tumours in the nostrils, or hypertrophy of the turbinated bones; constriction of the laryngeal orifice; tumours on the posterior nares, falling into the glottis and causing intermittent roaring; distension of the guttural pouches; tumours in the thorax; disease of the lymphatic glands of the pharynx; injury or disease of the trachea, or, finally, any distortion of the larynx.

The roarer generally has a characteristic cough in addition to the abnormal inspiratory sound. This cough is a loud, harsh, and dry sound.

Most roarers are also grunters in addition.

In testing a horse for its wind, it is customary to place him against a wall and make a feint to strike him. If he grunts he is further examined; if not, he is made to cough by compressing the larynx, and if the cough enforced sound healthy, he is passed. It is, however, best to have the horse galloped, and let him finish his run as he is going uphill. In the case of a draught-horse, the animal may be made to draw a load at a fair pace up an inclined plane, when, if he be a roarer, the characteristic sound will be made. All forms of roaring constitute unsoundness, yet in some cases of recent inflammation of the respiratory organs, the animal may be again examined after a reasonable interval before being finally rejected.

Treatment of Roaring.—In cases of abnormal growths in the nasal chambers, a careful examination should be made with the speculum and by percussion. If a polypus be detected, we may endeavour to remove it if operation is feasible. Similarly, if any removable adventitious growth be found to be the cause of the infirmity, it should be treated according to the requisite surgical methods.

When roaring follows influenza, catarrh, or strangles, it should be carefully treated. The animal should have light work only, and nutritious diet. Potassium iodide, with quinine and liquor arsenicalis may be given in moderate doses twice daily. In addition, the throat should be smartly blistered with ointment of cantharides and biniodide of mercury.

The greater number of cases of roaring met with however, are due to atrophy and paralysis of the laryngeal muscles.

Many methods of treatment have been suggested for these, which are the most hopeless cases. Blisters or the actual cautery have, however, succeeded in arresting the atrophic change when applied in the early stages to the skin of the laryngeal region.

Potassium chlorate is recommended as worthy of trial for arresting the fatty degeneration. In very bad cases the sound may be lessened by pads attached to the bridle and fitted carefully over the nostrils, so as to regulate the amount of air entering the larynx. If this method is ineffectual, tracheotomy may be performed, and the tube kept in during the rest of the life of the animal.

Probably the galvanic current is of great benefit, and is well worthy of careful trial.

Removal of the left arytenoid cartilage has been recommended by Herr Günther, but the cases thus operated on in this country are not sufficiently successful to warrant the general adoption of this method. Bad roarers can be used for slow work; they thrive badly as a rule, and often fall victims to slight disorders.

ACUTE CATARRH.

Synonyms.—Catarrh; coryza; common cold.

Definition.—By the term 'catarrh' we signify a condition characterized by inflammation of the mucous membrane of the nasal chambers, and of its continuation along the upper portions of the larynx and trachea. In this condition there is a discharge from the nose, and occasionally cough and sore throat.

Febrile symptoms are also often present.

Etiology.—Sudden variation in the temperature; ex-

posure to cold and damp; hot, badly-ventilated stables, and contact with affected animals, are among the chief causes of catarrh. Young animals, when first brought up into warm stables, are especially subject to attack. During the change of the coat there is also great predisposition to catch cold.

Symptoms.—Sneezing; redness and dryness of the Schneiderian membrane, followed by discharge, at first thin, but soon becoming turbid, yellowish-white, and profuse. There is also redness of the conjunctival membrane, with discharge of tears and drooping of the head. Febrile symptoms are sometimes manifested, and vary much in intensity. The internal temperature may rise about 3°, or even a little higher. The pulse and respiration are then accelerated, and the appetite is impaired. Debility and general dulness frequently supervene.

Prognosis.—Very favourable.

Treatment.—In mild cases rest from work, in a well ventilated but not draughty or cold loose box, will suffice, with attention to the diet. In all cases where the febrile manifestations are at all severe, febrifuges, as acetate of ammonium, may be given with nitrate or chlorate of potash twice or three times daily.

When the bowels are much confined, enemas of warm water may be administered, and, if necessary, two drachms of aloes may be given.

If the throat be sore and the cough troublesome, we may administer the compound tincture of camphor, or other remedies, in addition to the fever medicine.

In the early stages, while the mucous membrane is dry, inhalation of hot water vapour is useful in relieving the irritation. When the throat symptoms are severe, compound liniment of camphor, or liniment of turpentine, or cantharides should be applied externally. The diet should

be laxative, consisting of scalded oats, oatmeal, or linseed gruel and green food.

CHRONIC NASAL CATARRH.

Definition.—A discharge of varying character from the nasal chambers, which may be continuous or irregular.

Etiology.—Most of the cases of nasal catarrh are due to unhealthy condition of the mucous membrane of the nasal chambers or of the mucous cavities in connection with them, and are the result of protracted and severe cases of acute catarrh. It may arise from other causes, as external injuries, caries of the upper molars, disease of the superior maxillary bone, alveolar abscesses, disease of the facial bones, etc. These latter will be considered under 'Surgery.'

Symptoms.—There is a discharge of a glairy purulent fluid, and the Schneiderian membrane is of a leaden hue, or it may be blanched and thickened with infiltration.

The general health is generally somewhat impaired.

Treatment.—The animal should be rested, and liberal diet allowed. Salts of iron or arsenic are recommended internally, with or without vegetable tonics.

Locally, lotions consisting of four or five grains of sulphocarbolate of zinc, or half a drachm of sulphate of copper to the ounce of water, may be injected up the nostrils by means of an enema syringe or through a Rey's nasal funnel. The use of atomised solids is in some instances to be preferred to lotions.

Iodoform, blown into the nostrils by an insufflator, is an efficacious remedy.

DISEASES OF THE LARYNX.

ACUTE LARYNGITIS.—Definition.—Is an inflammation of the lining membrane of the larynx.

Two forms of acute laryngitis may be distinguished; acute catarrhal laryngitis, and œdematous laryngitis.

ACUTE CATARRHAL LARYNGITIS.—This is the simplest form, and is dependent upon the same causes as common catarrh, of which, indeed, it is usually one of the earliest and most prominent symptoms. There is pain and difficulty in swallowing, and on manipulation of the throat. Usually there is cough; at first hard and sonorous, but afterwards becoming less resonant.

There is swelling in the submaxillary space and over the parotid region. Nasal discharge may or may not be an accompaniment, although it is a constant symptom when catarrh is also present.

Febrile symptoms are more pronounced than they are in simple catarrh. There is diminution or loss of appetite, elevated temperature, and acceleration of pulse.

ŒDEMATOUS LARYNGITIS.—The structural changes in this form of laryngitis are distinctive. There is excessive infiltration of the submucous tissues of the pharynx and larynx, and the mucous membrane is pulpy and of a leaden metallic hue. In some instances patches of ecchymosis and gangrenous spots are met with on the inflamed membrane. The rapidity with which the infiltration of the laryngeal and neighbouring structures takes place, the suddenness of the onset, and its frequently fatal termination, are distinguishing features of this form of laryngitis. Œdematous laryngitis may succeed what at first appeared as an ordinary case of the catarrhal form, or it may begin suddenly and

run a very rapid course. It has also been met with as the result of the inhalation of poisonous acrid vapours and hot air. Sometimes, in a few hours, the swelling and infiltration become very considerable and the breathing much disturbed. The pulse is accelerated, the temperature elevated, and the visible mucous membranes become of a purplish hue, owing to deficient aëration of the blood. The nose is protruded, the superior air-passages being thus made to approach as near as possible a horizontal line. The glottis is indeed sometimes so occluded through excessive infiltration of the structures bounding the orifice as to cause imminent danger to life from suffocation.

Rapid effusion into the submucous tissue and swelling of the mucous membrane is termed 'œdema glottidis.' In these cases the respiration becomes suddenly difficult, and the inspiratory act is especially prolonged, and accompanied with a peculiar harsh sound succeeded by a short expiration. The alæ of the nostrils are dilated to their full extent, the face has an anxious expression, and there is great distress. The extremities are cold, and sweats bedew the body. The animal stamps with its feet, and the distress becomes increased; the visible mucous membranes become more livid, prostration ensues, and the horse, unless relieved, soon succumbs.

Sequelæ of Laryngitis.—Thickening of the mucous membrane; atrophy and degeneration of the laryngeal muscles; and ulceration of the structures bounding the rima glottidis.

Treatment of Laryngitis.—In severe cases the treatment should be as prompt as possible. Inhalations of hot-water vapour, medicated or not, and hot fomentations should be applied, but if the general distress and difficulty in breathing continue, tracheotomy may be necessary in order to avert suffocation. The hot-water vapour may be generated

by pouring hot water over chopped hay or bran in a nose-bag, and may be medicated by the addition of chloroform or opium, or other drugs.

In mild cases, inhalations, fomentations to the throat, succeeded by stimulating liniments or blisters, are recommended. Febrifuges may be given internally. The diet should be soft and laxative, and the horse should be warmly clad and placed in a well ventilated airy loose box. When deglutition is very difficult it is necessary to give all medicines in the water or food.

Belladonna is recommended in the early stages and should be administered by placing the extract between the horse's teeth as an electuary. During recovery the diet should be as nutritious as possible, and vegetable and mineral tonics should be administered in order to combat the great prostration usually left in these cases.

DISEASES OF THE BRONCHI.

ACUTE CATARRHAL BRONCHITIS.—Definition.—Bronchitis is a catarrhal inflammation of the mucous membrane of the bronchial tubes. It may be limited to the trachea and large bronchial tubes (simple bronchitis), or it may extend to their ultimate ramifications (capillary bronchitis).

Etiology and Nature.—The causes are predisposing and exciting. The predisposing causes are debility, previous attacks of bronchial inflammation, and cardiac diseases of a chronic nature. The exciting causes are exposure to cold and damp, exhaustion, and irritation of the bronchial membrane by noxious vapours, or by the accidental entrance of fluids or solids into the bronchial tubes; epizootic influences in some instances, as observed in certain forms of influenza; and lastly, bronchitis as met with in certain

specific fevers and under various other conditions depending upon malhygienic conditions.

Varieties.—Acute Bronchitis may be grouped under the following varieties: (1) primary or idiopathic bronchitis, which may involve the larger air-tubes or may extend to their ultimate ramifications; (2) secondary bronchitis, or bronchitis co-existing with specific fevers and other diseases depending on an unhealthy condition of the system; (3) mechanical bronchitis, or bronchitis depending on noxious vapours, fluids, or solids, irritating the lining membrane of the tubes.

1. Symptoms of Acute Bronchitis of the Large and Small Tubes.—Acute bronchitis is usually ushered in with chilliness, malaise, and febrile symptoms, though in many cases these may be trivial.

When fully developed, besides the symptoms of ordinary catarrh, there is a frequent hard and sonorous cough, which becomes softer as exudation into the bronchi increases in the progress of the disease. The appetite is impaired and the horse is dull and dejected. The pulse is increased and is rather soft; the respirations are much accelerated, being relatively much higher than the number of pulse-beats. In many severe cases indeed they are as numerous as the pulse, and may even exceed it in number.

Physical Signs.—Generally there is no alteration in percussion in bronchitis, though there may be dulness over portions of the chest where the lung is collapsed, owing to obstruction to the passage of air in inspiration by inspissated mucus, and also when pneumonia or other complications are present. In some instances of acute bronchitis attended with much secretion, the percussion note is more resonant than usual, owing to distension of the lung-tissue with air. This condition sometimes proceeds to actual emphysema. The breathing-sounds are coarse and noisy.

In the early stages, when the large bronchi are specially affected, loud, sonorous rhonchi may be heard over the anterior part of the chest, and also behind the scapula, at the upper and middle thirds of the chest. If the smaller ramifications be involved, sibilant râles are heard; they are of higher pitch, and wheezing and hissing in character. The milder forms of bronchitis yield no physical signs of importance.

As the disease progresses, there is secretion of viscid mucus from the bronchial mucous membrane, and the sounds in consequence become moist, being modified by the passing of the air through the bubbles of mucus. In the large bronchi, where the sonorous rhonchi were heard, large crepitations or mucous râles, as they are sometimes called, are now audible in their stead. In the smaller tubes lesser crepitant sounds or smart sibilant râles are heard.

In cases where the deeper and more sonorous sounds are in excess of the high-pitched sibilant râles, the disease is mainly confined to the large bronchi, and the smaller ramifications are entirely free. The prognosis in such cases is more favourable.

When the smaller bronchi are especially affected, the expirations are much prolonged, and the prognosis is more serious.

In some cases one or several bronchi may be blocked up, and thus portions of the lung may collapse, and consolidation, atrophy, and pulmonary emphysema may be induced.

As the disease progresses there is expectoration of a scanty, ropy, tenacious mucus. The discharge escapes to some extent through the nose, but the greater part passes into the mouth, and is swallowed. In the later stages, however, a profuse discharge escapes through the nostrils. The cough becomes more violent and frequent than at first, but

gradually becomes less severe as the secreted mucus becomes thinner and clearer, and finally disappears.

Bronchitis of the larger tubes is not generally a fatal disease; but when affecting the smaller bronchial ramifications and pulmonary vesicles, especially if there be profuse discharge of yellowish matter, which blocks up and occludes the small tubes and air-cells, the prognosis is very serious.

Secondary Bronchitis.—Is sometimes met with in rheumatism, glanders, 'and in young horses suffering from scrofulous arthritis it is a common complication, and is here found in conjunction with pulmonary embolism' (Robertson).

In these cases the bronchial symptoms are usually manifested during the progress of the primary disease.

Mechanical Bronchitis.—The symptoms of this form of bronchitis vary much with the nature of the cause, and are similar to those of acute catarrhal bronchitis. Mechanical bronchitis is induced by some irritant in contact with the lining membrane of the tubes. The irritating agent may be gaseous, as, for instance, smoke from burning buildings or acrid fumes; or it may be fluid or solid, as, for instance, food or other matters finding their way into the air-tubes. Mechanical irritation in some instances causes the condition known as plastic bronchitis, in which laminated fibrinous casts are formed in the air-tubes, and block up the lumina, thus inducing serious pulmonary mischief.

Morbid Anatomy of Bronchitis.—Inflammation of the bronchial tubes is attended with changes in the epithelial covering, glandular secretions, and in the subjacent tissues.

The mucous membrane is congested, sometimes intensely so, and undergoes more or less infiltration and thickening. It may acquire a granular aspect and soft pulpy consistence. The inflammation may be limited to the surface of the mucous membrane, but often pervades the sub-mucous tissues, in

some cases involving the whole thickness of the bronchial walls, and leading to infiltration and induration of the connective-tissue surrounding them.

In some instances the inflammatory process ends in ulceration, and ulcerated patches of varying size may be observed after death.

When the small tubes are especially involved, the thickening of the mucous membrane encroaches more seriously on the channels of the affected tubes, and their secretions tend to accumulate in them, and block up their lumina entirely.

In cases where the secretion is abundant or viscid, diffuse or circumscribed patches of collapsed or condensed lung tissue of varying extent are of frequent occurrence.

This condition (atelectasis) results from the plugging of the bronchi with inspissated secretion or pus. These patches may be seen after death on the surface of the lung, as depressed areas of a dark violet hue, the surrounding parts being of a light pink colour, and either healthy or emphysematous.

The collapsed portions are less crepitant than normal lung, but not so much as hepatized lung, and they resemble fœtal lung, sinking slowly in water.

On cutting into the lungs, the large and small tubes, and sometimes the trachea, contain a quantity of secretion. This condition, as well as the collapse, is usually limited to the anterior lobes of the lung, and rarely affects the large lobules except by extension. The fluid in the tubes in the earlier stage, after that of arrested secretion, is thin and serous, but afterwards becomes thick and yellow. In the trachea it is frothy, more or less, and is abundant in the small tubes, from which yellow matter may be expressed.

If a portion of the collapsed tissue be cut into, opaque serous blood-stained fluid, sometimes intermingled with purulent secretion, exudes from the cut surface. After a

time, the collapsed lung-tissue becomes atrophied, and replaced by fibrous material, and compensatory vesicular emphysema of the neighbouring lung-tissue is developed.

Pulmonary collapse is more generally met with in cases of secondary bronchitis and where the animal is in a very debilitated condition.

In all cases of bronchitis the bronchial glands are enlarged, and become more or less friable, and in the later stages still larger, owing to the accumulation of inflammatory products.

At an early stage in acute bronchitis the epithelium of the tubes is shed, and is not reproduced until after subsidence of inflammation, and the mucous membrane is thus left in an exposed condition. This shedding of the epithelium is probably due to infiltration of the basement membrane of the tubes. Bronchitis may end fatally by absorption of putrescent catarrhal products, by gangrene of the collapsed lung, or by sudden and copious effusion into the bronchi.

Treatment of Bronchitis.—The horse should be warmly clad, and placed in a well-ventilated loose box, and the diet should be liberal, nutritious and laxative.

It very rarely happens that we see a case of bronchitis in its earliest stage, in which anodynes, as, for instance, opium in moderate doses, are likely to be efficacious in reducing the irritation. After the early stage is passed, it is our object to promote the natural course of the inflammation, as we can no longer expect to cut short the attack. With this object in view the animal should be made to inhale hot-water vapour, which may be medicated with camphor or carbolic acid. In all cases of acute bronchitis, inhalation is of the greatest importance, but especially when the discharge is very profuse, is it of special efficacy in promoting the discharge from the bronchial tubes. If there be any laryngeal inflammation, stimulating embrocations, as, for

instance, compound camphor liniment, should be applied externally. If the bowels are inactive, enemas should be administered, and if they still continue very constipated, a dose of linseed oil may be given.

A draught, made up of three ounces of solution of acetate of ammonium, half an ounce of nitric ether, one drachm of camphor, and half an ounce of tincture of squills, may be given three times daily, in eight ounces of water.

In cases where the small tubes are much affected, blisters or stimulating liniments may be applied with advantage to the sides of the chest.

Where the cough is troublesome after the abatement of the acute symptoms, a draught of camphor and hyoscyamus, with a small dose of morphia, may be administered twice daily.

In the later stages, if there be much debility, vegetable and mineral tonics, as, for instance, sulphate and carbonate of iron, quinine, nux vomica, and gentian, are indicated, and if there be still much cough, camphor may be added.

Chronic Bronchitis.—Chronic bronchitis is met with in the horse either as a sequel of the acute form or as an independent disease.

It differs from the acute form in its slower progress, and in its symptoms being less severe, and is characterized by a persistent hard sonorous cough, and by the absence of febrile manifestations, and, moreover, the matter expectorated is rarely of that viscid cellular nature of acute bronchitis.

The sounds heard on listening to the chest are somewhat similar to those of the former, and the respiration is almost invariably embarrassed, especially when the horse is undergoing any severe exertion.

In some instances an attack of acute bronchitis may supervene on the chronic disease from very slight causes.

Chronic bronchitis seems to be in a great measure due to

thickening of the walls of the small bronchial tubes, narrowing and constricting their lumina.

In many instances this disease causes gradual loss of flesh, diminution of appetite, and general debility. When it occurs as an independent affection it is generally gradual in its onset and development, and of a very persistent nature when once established.

Treatment.—The diet should be liberal and nutritious, Rest is not necessary, though severe exertion should be prohibited. The remedies recommended in the acute form may be with advantage tried in the more persistent and chronic form of the malady.

Camphor, squills, hyoscyamus, digitalis, morphia, extract of belladonna, and hydrocyanic acid are recommended, and should be followed up by the administration of mineral or vegetable tonics or both. Among the latter, nux vomica, quinine, iron, and arsenic are well worthy of trial.

DISEASES OF THE LUNGS.

CONGESTION OF THE LUNGS.

Synonyms. — Pulmonary congestion. Pulmonary apoplexy.

Definition.—Is a hyperæmic condition of the lungs, dependent on engorgement of the pulmonary vessels with blood.

Etiology and Nature.—Congestion of the lungs is met with in the horse, not only during the progress of many diseases, such as pneumonia, laminitis, traumatic arthritis, enteritis, cardiac affections, and some epizootic fevers, but also as a distinct and independent affection.

Pulmonary congestion is acute (arterial) or passive (mechanical or venous).

Acute Congestion is the more frequent of the two forms in the horse, and is the more easily recognised. When accompanied by hæmorrhage from rupture of the pulmonary capillaries, it is sometimes spoken of as pulmonary apoplexy.

Acute congestion is the result of sudden exertion in an untrained condition, exhaustion, badly-ventilated stables, and various other causes which under some conditions induce this malady, but under other circumstances are provocative of bronchial or common catarrh.

Passive Congestion may be due to retarded venous circulation, as in mitral and some other cardiac diseases, or to general exhaustion, resulting in some fevers, and other debilitating affections.

When an animal in an untrained condition is suddenly called upon to perform any unusual exertion, the cardiac, pulmonary and muscular systems may not be able to respond to the increased strain put upon them. Under these circumstances—as, for instance, when an unprepared horse is suddenly put into the hunting-field—the heart's action becomes embarrassed and tumultuous, the blood accumulates in the pulmonary capillaries, and the breathing becomes more and more distressed, until, at length, he may die of suffocation, consequent on overloading and engorgement of the lungs with deoxidized blood. Under careful training the cardiac and other systems are gradually accustomed, by regular and careful exercise, to perform additional work, and their tone and vigour is enhanced, and the system responds duly to even severe strains.

Symptoms.—The symptoms of the acute forms of pulmonary congestion are in most instances of a very severe type, and the affection is specially characterized by very great dyspnœa. The horse stands with his limbs outstretched, and gasps for breath. All the available ordinary and extraordinary muscles of respiration are called into

play; the nostrils open and close in quick succession, and the flanks heave to and fro with great rapidity. Cold sweats bedew the surface of the body; the extremities become icy cold, and the visible mucous membranes show by their livid hue the deoxidized state of the blood.

The pulse is much accelerated, and may reach 100-140 per minute; it is feeble, oppressed, indistinct, and becomes almost imperceptible in severe cases, and there is a tremor all over the body.

The heart's action, which is irregular and tumultuous from the first, becomes still more embarrassed; the lungs become more engorged; and the breathing still more distressed, until, at length, death results from asphyxia or suffocation. In more favourable instances, however, the engorgement subsides, the heart regains power, the circulation through the lungs is restored, and the animal soon regains its normal condition.

In some instances frothy blood is discharged through the nostrils, from rupture of the engorged capillaries of the lungs; in others, this hæmoptysis, or discharge of blood from the lungs, does not occur. Auscultation in some cases reveals abundant minute crepitation, generally diffused, but sometimes heard only over limited areas.

It is unnecessary to say much of **passive pulmonary congestion**, as this is treated of under the various diseases of which it is an accompaniment.

It will suffice to mention, that although the symptoms in these cases are less marked, yet the disease is more frequently fatal than in the acute form occurring as the result of exertion in an unfit condition to bear the strain.

Prognosis.—Is favourable, except in very severe cases.

Sequelæ.—Acute congestion of the lungs is very liable to recur for some time after apparent recovery, and is not unfrequently followed by acute inflammation of the lungs.

Morbid Anatomy.—The pathological lesions found after death from acute congestion of the lungs, are as characteristic as the symptoms during life.

The lungs are found to be of a dark purple hue, firm and crepitant, and the vessels of the pulmonary tissue are engorged with deoxidized blood of a tarry consistence. Here and there darker spots may be seen, where extravasation has occurred into the parenchyma of the lung. The appearance of the lungs resembles that of the spleen; and the term splenification, or splenization, has thus been given to this congested condition of the pulmonary structures. The organs are more condensed and heavy than normal, but will generally float in water, thus differing from the condition which we shall shortly consider, termed hepatization, the result of inflammation of the lungs.

The lining membrane of the whole respiratory tract is congested, especially in the bronchial tubes, and is covered with frothy mucous secretion.

Both sides of the heart are filled with darkly coloured, thick, but not coagulated, blood. The right auricle and ventricle, with the large vessels in connection with them, are especially engorged.

In cases of passive congestion, associated with other diseases which have a fatal termination, the lungs are found to be more crepitant, and not so dark in colour, and there is a greater quantity of frothy mucus in the air-tubes.

Instances of death from acute congestion have not unfrequently occurred within a few days of purchase of a horse, and the lesions found after death have been ascribed by the uninformed to long-standing disease of the lungs. On some occasions the seller has, in consequence, been compelled to pay full value of the animal. And yet these very features, which were attributed to old-standing disease, are,

on the contrary, the characteristic results of acute disease. Indeed, the darkly coloured friable condition of the lungs, with the tendency to putrefaction and liquidity of the pulmonary tissue, without the formation of organized exudation, so far from being the result of chronic disease, are in all cases characteristic of acuteness of attack.

Treatment.—In the treatment of acute congestion, it is imperative that the horse have a plentiful supply of pure fresh air, and strict quietude and repose should be enjoined in all cases. The body should be well rubbed with wisps of straw and afterwards warmly clad. The legs should be carefully bandaged with thick wool or flannel, being pre viously rubbed with some stimulating liniment.

Internally, diffusible stimulants, as ether or alcohol, in its various forms, in moderate and frequently repeated doses, are of great value in helping to restore the flagging circulation. Acetate of ammonium, or nitrate of potash, may with advantage be given in addition to the stimulant.

Professor Williams speaks favourably of the tincture of arnica; but we have not found the efficacy of this medicine much to be relied upon in this disease.

If the symptoms are still unrelieved after this treatment, bleeding is necessary, in order to relieve the pulmonary congestion and the engorgement of the great veins and right side of the heart, and thus avert impending suffocation.

From three to four quarts of blood may be withdrawn. It is not advisable to repeat the bleeding, nor to apply mustard or other irritants to the sides of the animal, as these measures merely annoy the animal and thus increase the respirations and enhance the dyspnœa.

In order to guard against inflammation of the lungs, which sometimes succeeds acute congestion, as well as to prevent the recurrence of the congestion itself, careful management is required for some time after the abatement

of the acute symptoms. The diet should be light and nutritious, and easily assimilable. The box in which the horse is placed should be well ventilated, but not draughty, and the body should be kept warm with clothing. Water may be allowed freely from the first onset of the disease.

PNEUMONIA, OR INFLAMMATION OF THE LUNGS.

Inflammatory processes in the lungs comprise the following three chief varieties :

1. Acute, plastic, lobar, croupous pneumonia.
2. Catarrhal, lobular or broncho-pneumonia.
3. Chronic, or interstitial pneumonia.

ACUTE PNEUMONIA is now accepted as a general disease, of which pneumonic consolidation is the prominent local lesion. There is some probability, from its clinical features, of this disease being due to a living specific germ or bacterium.

CATARRHAL PNEUMONIA is inflammation of the lung-tissue associated with and usually secondary to inflammation of the bronchial mucous membrane occurring independently or as a complication of some specific fever.

It is at first usually limited to scattered groups of air-cells, and hence is termed lobular.

INTERSTITIAL PNEUMONIA.—Probably never occurs as a primary and independent affection. In all cases it is probably the sequel of some more acute inflammation of the pulmonary or bronchial tissues, or of the pleura.

ETIOLOGY OF PNEUMONIA.—The usual exciting causes of pneumonia are sudden chills, exposure to wet and cold, especially after severe exertion or fatigue, and confinement in draughty or foul and badly ventilated stables.

Pneumonia is especially prevalent during spring and autumn, when sudden changes in the atmospheric conditions are of frequent occurrence. Congestion of the lungs, and also previous inflammation of these organs, are both predisposing causes of pneumonia.

Pneumonia may also be induced by irritating gases, by the smoke of burning straw or wood, or other materials, by medicines, especially such irritants as ammonia, and by any foreign bodies which find their way into the lungs. It may result from injury to the lung through wounds in the chest-walls, or by fractured ribs penetrating into the lung-tissue; but pneumonia from these causes is not of such frequent occurrence as might be anticipated.

Pneumonia is met with in some specific fevers, as influenza, purpura, anthrax, and some others.

In pyæmia and glanders it is sometimes developed as the result of infecting material carried to the lungs.

Lastly, catarrh of the ordinary type, when neglected or treated carelessly, often terminates in secondary pneumonia by spreading of the inflammation downwards into the pulmonary vesicles.

PATHOLOGICAL CHANGES IN PNEUMONIA.—Inflammation of the lungs generally commences in the inferior portions, and invades the tissues from below upwards (Williams), and in all cases the small bronchial tubes are more or less involved in the process.

The pneumonia process not unfrequently involves both lungs, especially when associated with or preceded by bronchial inflammation, but far more often it affects one side only, the right side being oftener involved than the left.

1. PATHOLOGICAL CHANGES IN ACUTE PNEUMONIA.—The local changes in acute pneumonia consist in intense inflammatory hyperæmia of the lungs, and in the

exudation of a large quantity of coagulable material into the tissue.

Acute inflammation of the lungs is termed croupous, from its resemblance to the pathological changes resulting in croup.

It is also termed lobar, because it almost always invades a large part of the lung.

There are three stages in this inflammatory process, but it must not be imagined that each one occurs simultaneously throughout the whole of the affected portion of the lung. On the contrary, the changes proceed irregularly, and while one portion is in the second stage another may be in the third, thus accounting for the marbled appearance of consolidated lung (Green).

The stages are firstly that of **engorgement**, secondly, that of **red hepatization**, and thirdly, that of **grey hepatization.**

In **the first stage** the lung becomes very vascular, and the changes in the vessels and circulation are such as already mentioned under 'Inflammation.' The absolute weight and specific gravity of the lungs are increased, their elasticity is diminished, and they are less crepitant and more friable than normally. They are of a dark red colour, and pit on pressure, and the cut surface yields a red, frothy, tenacious, viscid liquid.

In **the second stage**, that of red hepatization, there is exudation of liquor sanguinis and migration of blood-cells into the pulmonary tissue. The exuded liquids coagulate in the vesicles and terminal bronchioles, and the coagulum contains many white and some red blood-cells.

The lung is of a dark reddish hue, often passing into grey. It is much heavier than in the first stage, and is increased in size, so as to be often marked by the ribs.

It is quite solid, sinks in water, and cannot be artificially

inflated; does not crepitate under the fingers, and is very friable, breaking down readily with a soft fracture. The cut surface has a distinctly granular appearance, seen especially when the lung-tissue is torn. This appearance is due to the coagulated matter which plugs up the alveoli.

During this stage there appears to be but little alteration in the walls of the vesicles, or in the epithelium lining them; the latter, however, is usually swollen and granular, and the alveolar walls often have a few leucocytes adhering to them. The pleura covering the solid lung is always more or less inflamed, and is opaque and coated with an exudation of lymph.

In **the third stage**, or that of grey hepatization, there is continuance of the emigration of the white cells, which escape from the vessels and gradually accumulate in the pulmonary alveoli. The epithelial cells lining the alveolar walls become more swollen and granular, and their air-vesicles become filled with cells, so that the fibrous exudation is no longer visible as an independent material, as in the second stage.

The cells soon undergo rapid fatty changes, and the fibrinous material breaks down. Thus the alveoli as usually seen are filled with granular elements, and their walls, though usually unaltered, may be partly disintegrated. The weight, density, and friability of the lung now become greater than in the stage of red hepatization, although the granular aspect of the cut surface is much less marked.

The lung-tissue is soft and pulpy, and a purulent fluid exudes from the cut surface.

The lung itself changes in colour from a dark red-brown to a grey or yellowish-white.

Terminations of Acute Pulmonary Inflammation:—

(A.) Resolution.—This is by far the most common and favourable termination of croupous pneumonia.

(B.) **Abscess.**—This is a rare termination. When one or more abscesses are formed they may discharge themselves by the air-tubes, or may undergo caseous or calcareous degeneration.

(C.) **Gangrene.**—This is a still rarer termination. It is due to extensive formation of coagula in the pulmonary and bronchial vessels, together with considerable hæmorrhage into the pulmonary tissue, and is predisposed to by malhygienic conditions to which the animal may have been exposed.

(D.) **Chronic Pneumonia.**—If the inflammation do not subside, and the exuded liquid become absorbed, the alveolar walls gradually become involved, and thickened by fibro-nucleated growth.

2. **PATHOLOGICAL CHANGES IN CATARRHAL OR BRONCHO-PNEUMONIA.**—The inflammation of the pulmonary tissue is in this form always associated with catarrh of the bronchi.

Broncho-pneumonia may be caused firstly by the collapse of the lung-tissues from inflammation of the bronchial mucous membrane; secondly, by direct extension of the inflammation from the bronchi into the air-cells; and lastly, the inflammation may originate in the air-vesicles themselves before the bronchi are attacked. In catarrhal pneumonia, the inflammatory products which fill the alveoli consist very largely of cells derived from the epithelium of the alveoli and from the bronchial mucous membrane.

Exudation and emigration play a much less prominent part in the process than they do in croupous pneumonia.

This form has been termed catarrhal pneumonia, from the preponderance of epithelial products, and from the association of the pulmonary with the bronchial inflammation.

Resolution is less readily effected in broncho-pneumonia

than in croupous pneumonia, and is sometimes so much prolonged that thickening of the bronchi and alveolar walls remain.

3. PATHOLOGICAL CHANGES IN INTERSTITIAL OR CHRONIC PNEUMONIA.—This form, like all other chronic inflammations, is characterized by gradual increase in the connective-tissues of the organs, leading to induration of the textures and obliteration of the alveolar cavities. It is often associated with catarrh and dilatation of the bronchi, and sometimes with ulceration of the bronchial walls.

The thickening of the connective-tissue has been observed in the pneumonia of glanders, in which the exuded materials break down into a thin purulent fluid.

SYMPTOMS AND COURSE OF PNEUMONIA.—Inflammation of the lungs in the horse is mostly associated with bronchitis and pleurisy, though uncomplicated cases of true acute pneumonia do sometimes occur.

In many instances, the first manifestation of this disease is the catarrh which accompanies or precedes the inflammation of the lungs. Pneumonia is sometimes ushered in with a severe rigor, succeeded by a hot stage. The pulse is accelerated but very variable, and numbers usually from 90 to 100 beats per minute. The artery is hard and full at the outset, or it may be soft and full. In some instances it is irregular, intermittent, or dicrotic.

The temperature is raised and may read from 103° F. to as high as 106°. The skin and extremities are cold, and the visible mucous membranes are red and injected. The bowels are constipated, and the fæces coated with mucus.

The horse loses his appetite, and wanders to and fro in his box in a dull, dejected manner, showing no inclination to lie down.

If the case be complicated with pleurisy, the breathing

is more painful and abdominal; but in pneumonia not associated with pleurisy there are no signs of pain.

There may be dull dry cough, but there is seldom much expectoration, though rusty or blood-stained, more or less viscid tenacious matter, is sometimes discharged through the nostrils in croupous pneumonia, whereas in bronchitis and catarrhal pneumonia the discharge is more or less purulent. The urine is lessened in amount, and there is excess of urea and diminution of salines, especially chlorides, which probably, as in man, reappear in the urine during convalescence in full amount.

As the inflammation progresses the respirations, which at first were but slightly accelerated, gradually become more so, until the crisis, when they are much quickened. The breathing is also sometimes much accelerated in the congestive paroxysms, which are not at all infrequent during the progress of the malady. The febrile symptoms extend over a period of several days, or even longer, and then subside.

Some authorities describe the temperature as gradually subsiding; but we have noted very many instances of acute pneumonia where the *defervescence* of the fever has occurred suddenly, the temperature falling by *crisis* from 105° or more to near the normal in the course of two or three hours. The pulse falls, the appetite returns, the secretion of urine is again abundant, and the animal is soon convalescent.

Physical Signs.—Percussion reveals increased dulness in the second and third stages of ordinary acute pneumonia; there is, however, usually no impairment of resonance in the first stage.

In the early stages the respiratory murmur heard in auscultating the chest is harsher and louder than normally; and abundant fine crepitations are heard during inspiration, and sometimes also in expiration.

In the second stage the respiratory murmur is replaced by the *very characteristic tubular or bronchial breathing.* In some instances there is a total absence of breathing sounds in this stage, and the fine crepitations are not heard.

In the third stage of an ordinary attack ending in resolution, the bronchial breathing gradually gives place to crepitation of a moist coarse character, termed redux, or secondary crepitation, and this gradually passes into the ordinary bronchitic râles.

Pleural friction may be heard in addition to the sounds due to the pneumonia, when this disease is complicated with inflammation of the pleural membranes.

Acute pneumonia, especially when associated with bronchitis, is said to be sometimes complicated with laminitis, or inflammation of the vascular structures of the feet.

When pulmonary inflammation occurs as a **secondary affection,** especially when associated with some specific fever, or when the animal has been subjected to malhygienic and debilitating conditions, the attack is very apt to assume the adynamic or asthenic type, and is more likely to end in breaking down of the pulmonary tissues than in resolution.

Under such circumstances the pulmonary inflammation is more of the catarrhal or lobular type, which is chiefly characterized by filling of the alveoli with cells derived from the epithelium lining them, and by the fact that exudation and emigration play a much less prominent part in the process than they do in croupous pneumonia. Catarrhal pneumonia terminates by gradual defervescence of the febrile symptoms, or lysis, as it is termed.

Prognosis.—Is favourable in most instances. Pneumonia may prove fatal from the intensity of the fever in the early stages, and also during the stage of hepatization, from suffocation occurring as the result of the pathological changes. In double pneumonia, or inflammation of both

lungs, the prognosis must be very guarded, as it is far more frequently attended with a fatal result than is the single form.

Secondary pneumonia (*ceteris paribus*) is more serious than inflammation of the lungs occurring as a primary affection.

Treatment.—In the treatment of pneumonia, concerning which there is so much divergence of opinion, it is above all things necessary to consider the form of pulmonary inflammation in each individual case; the constitution of our patient; and, finally, the condition, work, diet, and hygienic surroundings under which he has been placed. The animal should be placed in a well-ventilated, but not draughty, loose box. This is one of the first essentials in the treatment of pneumonia, as impure air, containing organic germs, is apt to cause the inflammation to take on a suppurative form. If possible, the temperature of the box should be kept at 70° to 75° F.

The body should be clothed with rugs, and should be gently rubbed down occasionally with wisps of hay.

Bleeding should be practised only under exceptional circumstances. For example, in treating heavy draught horses kept in very high condition, as many of the agricultural and dray horses are, and also plethoric horses of other breeds, when the dyspnœa, or difficulty of breathing, is great, and the fever very high, it is our custom to remove from four to six quarts of blood. Under no circumstances do we repeat the operation.

Though practised formerly, and at the present day by some in almost all cases of acute lobar pneumonia, bleeding is not only of no advantage in many cases, but even positively harmful, and delays the convalescence of the horse, even though it may seem to relieve the dyspnœa for a time. In secondary pneumonia *bleeding should never be practised.*

We may allow the horse as much tepid or cold water, if he prefer it, as he will take; and with this object should leave a moderate supply by him.

The diet should be laxative and nutritious: bran-mashes, oatmeal gruel, linseed gruel, hay in moderate quantity, and roots or grass may be allowed. After the subsidence of the fever, stronger nutritious diet should be substituted.

During the fever, a draught containing five minims of Fleming's tincture of aconite, four ounces of liquor ammoniæ acetatis, one ounce of nitric ether, and two drachms of nitrate of potash, may be given every four hours, until the acute symptoms begin to abate. We entirely concur with those authorities who hold that tartar emetic, digitalis and calomel are of no effect in curtailing the duration of the pyrexia, and that opium should only be given when the pneumonia is associated with pleuritic pain. Owing to the irritable condition of the bowels in pneumonia, active purgatives should never be given. If there be great constipation, enemas or small doses of oil may be given.

In the later stages, when the pulse is weak and dicrotous, and from the earliest stages when the inflammation is of the adynamic or asthenic type, stimulants are indicated.

Six or eight ounces of whisky may be administered three times daily, and should be persevered with if it prove beneficial. If, on the contrary, the breathing becomes still more distressed, the treatment should be discontinued immediately. Bromide of ammonium and potassium are recommended by Williams as useful in moderate doses, having a calmative effect.

During convalescence vegetable and mineral tonics are indicated.

Finally, with regard to local applications:

When the extremities are icy cold they may be rubbed ith stimulating applications of a non-irritating kind.

Woollen cloths wrung out from hot water may be applied with great advantage around the chest, and should be renewed every two hours, or oftener, so long as the acute symptoms last.*

The indiscriminate use of blisters and sinapisms to the chest is a practice to be deprecated. But we are of opinion that in many cases of pneumonia decided advantage is to be obtained by vesication.

ASTHMA AND BROKEN WIND.

ASTHMA.—Definition.—Asthma is a morbid condition characterized by paroxysms of dyspnœa, probably depending upon spasm of the circular muscles of the bronchi, and often accompanied by a wheezing noise in the respiration.

Etiology and Nature.—The causes of asthma are heredity, direct and reflex nervous irritation, fatigue, overwork, and debility. Asthma bears a close resemblance in many points to broken wind, and appears to be due to spasm of the circular muscle-fibres of the bronchial tubes. If prolonged, asthma frequently terminates in that paralytic condition of the bronchial tubes, and in those changes in the structure of the pulmonary-tissue, constituting broken wind.

The latter condition may be aggravated by further spas-

* Great care is necessary in the mode of application of hot rugs, and several methods have been suggested. In the first place, the hot pack should be closely applied, so that no cold air can pass between the rugs and the skin. In the next place, the temperature of the rugs should be as high as the animal will bear. A good method of applying this treatment is to obtain a piece of felt about an inch thick and a foot and a half wide, fitted with straps. After being well wrung out from hot water and applied closely to the chest, a waterproof lined with flannel should be strapped round the felt. A simpler method is the application of an ordinary blanket, folded three or four times, over which is placed a dry rug, the whole being fastened with a surcingle or plough line.

modic attacks, which gradually involve more of the bronchial tubes.

Asthma is by many regarded as an early stage of broken wind, but as many cases undoubtedly recover without passing into the more serious condition, we have thought it advisable to consider it separately.

Symptoms.—Asthma is characterized by sudden spasmodic dyspnœa, which resembles that of broken wind in that the inspiration is easier than the expiration. The latter is usually of a jerky character, but has a less distinct double action than in broken wind.

In asthma the wheezing is more distinct; there is more exhaustion with less cough, which is not so hollow as in broken wind, but is short, quick, or suppressed. The suddenness of the paroxysms, their true spasmodic nature, their severity, their sudden accession and decline, and unaccountable disappearance, are marked features of asthma.

The febrile disturbance is severe when the disease is fully developed, and the chest is fixed, as it were, and there is increased movement of the abdominal muscles. When the cough is severe, small pellets of mucus are discharged through the nostrils.

If the chest be auscultated the wheezing noise is very distinctly audible in the anterior and lateral regions. The spasms may last a few days, or may extend over several weeks, and then disappear or pass imperceptibly into broken wind.

Treatment.—The diet should be of a laxative nature, and if the cough is very severe the sides of the chest may be blistered with great advantage.

The drugs of most value as internal remedies are digitalis, amyl nitrate, belladonna, lobelia, chloral hydrate, opium, and the bromides of potassium and ammonium.

Iodide of ethyl, used as an inhalation, is well worthy of trial.

BROKEN WIND.—Definition.—Is a nervous, inflammatory disease, characterized by difficult and spasmodic breathing, the inspiratory act being easily performed, the expiratory being very prolonged, and accomplished by two apparent efforts.

The dyspnœa is constant, and though marked by remissions and exacerbations, is not truly intermittent, as in asthma, and the cough, spoken of as the 'broken-winded cough,' is short and nervous.

Indigestion, flatulence and cardiac disease sometimes aggravate the dyspnœa.

Nature and Etiology.—The exact nature of broken wind is still a disputed point.

According to Professor Gamgee, broken wind is at first a purely nervous affection, dependent on an unhealthy condition of the digestive organs, and the pathological lesions are to be attributed to the effects of the nervous disease, which especially involves the pneumogastrics. He further attributes the condition of the digestive organs themselves to improper dieting, or to constitutional predisposition to indigestion and tympanitis.

With this view we entirely agree.

According to others, the manifestations of the disease are held to be *primarily* due to emphysema, and other structural changes in the lungs. Now, although pulmonary emphysema together with alteration in the tissue of the air-cells are found in very many instances of broken wind, it must nevertheless be borne in mind that these changes are by no means always present, for in some cases there is no evidence of emphysema or other structural pulmonary lesion after death.

And again, it is noteworthy that pulmonary emphysema,

especially of the vesicular form, has been found in many horses which have not at any time shown symptoms of broken wind.

Now, therefore, although it is probable that the amount of respiratory derangement bears a close relation to the extent of the emphysematous condition of the lungs, we can nevertheless only regard these lesions as the *occasional* result of some other diseased condition, and in no measure as the prime cause.

Regarding broken wind as dependent on disturbed innervation, traceable to derangement of the digestive system, the phenomena of this disease, and the variable pathological lesions found, may be readily accounted for.

Between the sub-epithelial mucosa and the submucosa of the small bronchial tubes there is a continuous layer of circular non-striped muscular tissue. This layer is especially conspicuous and important in the smaller microscopic bronchi (Klein), and by contracting aids the expulsion of tidal air from the lungs in expiration.

This muscle in broken wind is first spasmodically contracted, and then afterwards becomes paralyzed, and undergoes degenerative changes. This is owing to the irritation set up by indigestible food acting on the gastric branches of the pneumogastrics, and reflected through the pulmonary branches of the same nerves. This spasmodic contraction or paralysis arrests both the respiratory acts, especially the expiration, which becomes double, and requires a double contraction of the abdominal muscles to force out the air from the distended air-cells and now inactive bronchial tubes.

Round-chested horses sometimes become broken-winded without any apparent cause, and difficulty in expiration may be attributed in such cases to the limitation of the thoracic contraction. It will easily be seen that when the shape of

the chest is round the thoracic movements are more limited, and the horse will in consequence not be able to take deep inspirations or make strong expiratory efforts.

The nervous or functional derangement of the bronchial tubes and air-vesicles is soon succeeded by structural changes. The latter become inflated, and thus the circulation in the capillary blood-vessels is impeded, and the nutrition of the bronchial mucous membrane and muscles, and the walls of the vesicles themselves, becomes impaired (Williams).

Degeneration of these structures now follows, and the lungs become more and more emphysematous, the air-cells are dilated, their walls are stretched, and air now becomes intercalated among the connective-tissue of the lung.

In this manner vesicular and interlobular emphysema may frequently be produced. The right side of the heart may become hypertrophied and dilated in consequence of these changes in the lung structure.

The dyspnœa of broken wind seems to result from the emphysematous condition of the lung, as well as from general impairment of the contractile power of the pulmonary organs.

Having now reviewed the proximate causes of broken wind, we may now consider the more remote causes.

They are:

(1.) **Heredity.**—The offspring may inherit the same bodily conformation and temperament as their parents, and thus be more liable to become similarly affected.

(2.) **Dietetic.**—Defective dietetic conditions are largely responsible for the production of broken wind. As the diet of the coarser breed is frequently innutritious and bulky, and the animals often worked after a heavy meal with full allowance of water, they are more frequently

affected with this disease than better bred horses which are more carefully attended to.

Professor Robertson says that chopped hay is especially liable to induce broken wind, and has noticed in some districts that while the agricultural horses were fed on oats, with hay or oat straw, the fodder being given in full supply, broken wind was unknown, but that when the extensive use of chopped hay and oat-straw was adopted, this disease became comparatively common.

(3.) **Changes left by Previous Diseases.**—Those changes in the lung tissue which arrest the contractile power and action of pulmonary air-tubes may cause broken wind.

Symptoms.—The symptoms in confirmed cases of broken wind are unmistakable, but when not so fully developed the disease may be occasionally overlooked.

The inspiratory movement is performed rather quickly and with ease. The expiratory movement is much prolonged, difficult, and accomplished by two apparent efforts. It begins by a rapid contraction and upward movement of the posterior walls of the abdomen, and is suddenly stayed before the act is completed, and then continued slowly by an upward and forward movement of the abdominal muscles.

The cough of broken wind is characteristic, being short, of little force, and suppressed, and seems to be ejaculated with a kind of grunt through the upper part of the trachea (Blain). When the animal is in fair condition, the cough generally occurs only at long intervals, and rarely in paroxysms; but at the commencement of the disease, and when the horse is excited from any cause, or during exercise, it is apt to break out in paroxysms.

In many instances cough is one of the first indications of broken wind.

After feeding, the symptoms are more severe, and they

are liable to exacerbations from extremes of heat and cold, and other atmospheric changes.

The chest is rounder than it should be, and the thoracic movements are much impaired, while the movements of the abdomen are violently put into action during a paroxysm of cough.

In well-developed cases the loud sonorous wheezing noise may be distinctly heard while near the animal. If the chest be carefully auscultated, rhonchus and sibilus will be audible all over the chest, but the sounds vary much in character, even in the same animal, at different times.

The respiratory murmur is diminished or absent. In the inspiration a 'cooing rhonchus or friction sound' is heard; whereas a weak crepitus is audible in the prolonged expiration. Percussion reveals increased resonance in various parts of the chest. In confirmed cases the digestive organs are weak and easily deranged, the bowels are tympanitic, and borborygmi are frequent. The horse is debilitated and unthrifty, and the coat is often harsh, dry, and scurfy. When worked, he is easily fatigued, and perspires easily, and the bowels are generally loose.

Morbid Anatomy.—The pathological lesions found after death, which is in most cases due to some other disease or to accident, are very variable in character.

The organs in which the changes are most frequently met with are the lungs, heart, stomach, and intestines.

If death occurs during the early stages, no marked change may be found anywhere, though in some instances the lungs are slightly paler, and have greater buoyancy when floated in water than normal pulmonary tissue.

When the disease has existed for a long time, the usual pathological lesions which may be found in the lungs are interlobular and vesicular emphysema, thickening of the

walls of the small bronchi, hypertrophy of the mucous membrane of the larger tubes, and pallor of the pulmonary parenchyma.

Vesicular emphysema consists in permanent dilatation and enlargement of the infundibula and air-vesicles in larger or smaller areas of the lungs. As the process continues, communications are established between the adjacent groups of air-vesicles by atrophy and degeneration of their walls.

In this form of emphysema little vesicles containing air may be seen beneath the surface of the serous covering of the lung.

Interlobular emphysema is due to distension with air of the connective-tissue between the separate vesicles and the air-tubes.

In most cases of broken wind the heart, especially the right side, is hypertrophied and dilated, and is heavier and more bulky than normally.

The stomach is much dilated, and its walls stretched, and in old-standing cases similar changes may sometimes be found in the intestinal tract.

Treatment.—Above all things, it is essential that the diet should be carefully attended to. The food should be nutritious and digestible, and in moderate quantity, and the water-supply should be well regulated. The symptoms of broken wind may be ameliorated by all remedies which increase the general condition and improve the digestive powers.

Arsenic, in the form of Fowler's solution, which may be given in the drinking-water, is especially recommended. Nux vomica and vegetable bitters may be given in addition.

The arsenic may at first be given in moderate doses twice daily for two or three weeks, and then once a day for

a similar period, and then finally once every alternate day.

Purgatives in moderate doses, when occasionally administered, are beneficial in regulating the action of the bowels.

It is well known that some unprincipled persons adopt special measures in order to pass off a broken-winded animal for sale.

With this object they allow little or no food and water, and give the animal a sharp trot, in order that the bowels may be emptied.

They administer also various drugs, such as digitalis, opium, and other agents which have a temporary sedative effect, and in this manner have deceived many persons.

PLEURITIS, OF INFLAMMATION OF THE PLEURAL MEMBRANE.

Definition.—Pleurisy is inflammation of the visceral and parietal layer of the pleural membrane, and is termed primary (idiopathic), or secondary when depending upon some other disease of primary or general nature.

Etiology.—Pleurisy is due to

(1.) *Extension of the inflammatory action to the pleura, from contiguous or neighbouring parts.*

For example, pleurisy is frequently associated with, or supervenes on, pneumonia, bronchitis, and also cardiac and pericardiac diseases.

The pleural membranes are especially liable to be involved in this way when the primary disease is of an epizootic nature.

(2.) *Irritation from injury or morbid growths.*

Wounds penetrating the thoracic walls, or fractured ribs, are apt to set up inflammation of the pleural membrane.

The accumulation of fluid in the pleural sac, and the development of morbid growths in contact with the membrane, are also liable to set up pleurisy.

(3.) *Exposure to cold and damp and sudden atmospheric changes.*

(4.) *Certain enzootic and epizootic fevers and other constitutional diseases.*

For example, pleurisy is a frequent concomitant of some of the specific fevers, as, for example, influenza, and of constitutional diseases, as rheumatism, suppurative phlebitis and pyæmia.

Symptoms.—The fever of acute pleurisy is ushered in by a slight chill or by an actual rigor. The animal may be restless, and show signs of pain, aggravated by breathing, which, though accelerated, is performed carefully. The pain is increased when the horse is moved round, and the chest wall is very tender to pressure or when percussed, and the expression is anxious and indicative of great pain. The pulse is increased in frequency, but its ratio to the breathing is only slightly disturbed, and is hard and more incompressible than in pneumonia. The temperature is raised to 103° or 104°, the mouth is hot and dry, but the expired air is not so heated as in pneumonia.

In the inspiratory act the ribs are fixed, and in consequence of this there may be observed a furrow on the line of demarcation, called the pleuritic ridge. This extends from the bottom of the posterior part of the chest, and runs obliquely in an upward and backward direction to the anterior spine of the ilium. Cough is a frequent symptom of pleurisy. It is shorter and more painful than in pneumonia, and is attended with little or no expectoration.

If the chest be examined a friction sound, or 'dry leather creaking,' as it is sometimes termed, may be heard. This is owing to the rubbing of the two pleural surfaces together.

This sound appears as if close under the ear, and as the lung moves to and fro it is consequently double. It is not heard if the breathing cease, and the area over which it is audible may be very limited.

The friction may in many instances be felt by placing the hand over the involved area of the chest-wall. The vesicular murmur is weakened, and percussion in this dry stage reveals nothing.

The inflammation may now subside, with the effusion of very little lymph, in which case the friction sound passes away. If, on the other hand, the fluid gradually accumulates, in this case also this sound disappears, as the two roughened surfaces are separated by a layer of fluid.

When absorption of the fluid takes place, the two layers come together again, the sound is heard again, and is called 'redux friction.'

With the effusion of fluid the more active febrile symptoms and pain abate, the temperature may fall a little, the pulse is less jarring, and the pleuritic ridge less distinct.

If the effusion now accumulate in very large amount, the symptoms become more serious ; the pulse is increased to 80 beats or more per minute, and is of smaller volume, and may be irregular in rhythm and volume. The respirations become more laboured, and the difficulty of breathing may be very great ; the flanks heave, and there is flapping of the nostrils.

This dyspnœa is to be attributed to compression of the structure of the lung by the accumulated fluid, which, if not absorbed, is spoken of as dropsy of the chest, or **hydrothorax.**

The animal's head is generally protruded, and dropsical swellings appear in various parts. They are usually first noticed along the inferior part of the thorax, spreading along the floor of the abdomen, and may at length involve the limbs.

Prognosis.—Mild cases of pleurisy generally recover in a few days to a week. The more serious cases, however, where there is much effusion of lymph, are of longer duration, and though the prognosis is, in most cases, favourable, yet the changes in the pleural membrane, and the adhesions formed between the two layers, often continue for life.

Cases of pleurisy associated with specific influences are liable to be much protracted, or even prove fatal from the effused lymph becoming purulent. This condition is termed **empyæma.**

Morbid Anatomy.—The inflammatory process commences in hyperæmia of the pleural membrane. Exudation of fluid and blood-cells into the pleural cavity soon follow.

Gradually the surfaces become opaque, roughened, and very vascular, and covered over with a fibrinous layer, whilst more or less fluid transudes into the cavity.

The exudation from the vessels forms a soft, elastic membranous or reticulated coating, enclosing many cells in its meshes.

In this way the opposed surfaces of the pleural membrane may be glued together.

The exuded liquid varies considerably in amount, and is always turbid, in this respect differing from the non-inflammatory effusions.

It contains flakes and masses of coagulated fibrin, and many cells, which in the early stages are almost entirely emigrants.

If now the inflammation subsides, and the effused fluid is not sufficient to prevent the two surfaces coming into contact, they may become adherent, the union being effected by the formation of connective-tissue. If, however, the inflammation is severe, or there is much effusion, organization and adhesion cannot be effected.

The presence of much fluid interferes with its own

absorption by pressing upon and impeding the circulation in the blood-vessels and lymphatics, and thus the two surfaces are kept apart; but when the liquid is absorbed, the two surfaces may then grow together.

When the inflammatory action does not subside, or is of a very severe type, or depends upon some specific disease, pus may be formed and accumulate in the pleural sac. As the connective tissue becomes involved, granulation-tissue is formed, and may continue to generate pus, and discharge it into the pleural sac. The membranes become thickened, and the new tissue contracts in its organization, producing more or less retraction of the chest-wall. When the effusion has been very great, the pulmonary tissue is found much compressed, and when incised is tough, pale and dry.

The pleura of both sides may be involved, but usually the inflammation is confined to one, more frequently the right.

Treatment.—The horse should be placed in a loose box well ventilated, but of equable temperature, and should be warmly clothed with rugs. The diet should be at first nutritious, and such as is calculated to keep the bowels in regular action. As in pneumonia, bleeding is not recommended, except under very special circumstances. When the pleuritic attack is of an acute sthenic type, and not associated with any other constitutional disease, moderate bleeding in the early stages is recommended by some eminent authorities. The removal of from four to six quarts of blood under such circumstances is said to be followed by marked benefit and relief of the acute symptoms. We, however, do not practise bleeding under any circumstances in pleurisy, unless, indeed, the attack is associated with acute inflammation of the lungs. Internally, saline febrifuges may be administered, as in pneumonia, and if the pain be great, tincture of opium may be given in addition. Fleming's tincture of aconite is a very valuable remedy in the acute

stage, and may be given in five or six minim doses every four hours. In cases where the abdominal pain is great, some authorities recommend the injection of forty minims of solutio morphiæ hydrochloratis hypod. (B.P.) into the subcutaneous tissue at the point of the sternum.

Woollen cloths wrung out from hot water and applied round the chest, as in pneumonia,* are of great service in pleurisy also. They should be applied for about two hours continuously, and should be renewed four or five times daily, as long as the acute symptoms last.

If after abatement of the severe febrile symptoms the animal seems to make no progress towards recovery, but still breathes with difficulty, and the temperature remains high, stimulating liniments applied to the chest are recommended as very useful.

About four ounces of whisky, or other alcoholic liquor, may be given also under these circumstances three times daily, and if we have any reason to suspect the accumulation of liquid in the pleural sac, one drachm of potassium iodide may be given with the drinking-water two or three times a day.

In those cases where debility follows the acute symptoms, quinine and salts of iron may be given in addition to the above remedies.

When the effusion is extensive, and, although all therapeutic measures have been taken, remains unabated after three weeks or so, thus interfering seriously with the respiration, it is advisable to perform the operation of paracentesis thoracis, in order to relieve the pressure of the fluid on the lungs.

Some authorities have highly recommended cantharides in doses of three to six grains, with half a drachm of digitalis

* Vide 'Treatment of Acute Pneumonia,' page 162.

and two drachms of nitrate of potash in the form of a ball, given twice daily for a week.

But, presuming that after the administration of the therapeutic agents the effusion rather tends to increase than to become lessened in amount, we should not delay the operation. We may use the ordinary trochar and canula, or pneumatic aspirator, and should puncture the chest-wall in the eighth or ninth intercostal space at a point midway between the upper limit of the fluid and the bottom of the pleural cavity.

It is not necessary to remove all the fluid, as after the escape of a fair quantity the rest may become absorbed by the vessels, through which the circulation again passes freely.

If necessary, the operation may be repeated in a couple of days.

Tonics with nitric ether may be given after the operation, and the bowels should be regulated by laxative diet, which should be of a nutritious character, and if necessary, mild doses of oil, or other aperient, may be given in addition.

CHAPTER VI.

DISEASES OF THE CIRCULATORY SYSTEM.

PHYSICAL EXAMINATION OF THE HEART AND PERICARDIUM.

Diseases of the heart and pericardium, though of rare occurrence in the horse as compared with man, are nevertheless of great importance, and are by no means of unfrequent occurrence. In examining the heart, the methods of inspection, palpation and auscultation are adopted, as in the detection of diseases of the respiratory organs.

In the horse the heart is not so accessible for examination as in man, and in consequence 'of the ponderous shoulder closing in upon the keel-shaped thorax we are not able to distinguish the heart-sounds at different parts, and to determine the space filled by the organ, however large it may be' (Gamgee).

Thus the difficulties to be encountered in the detection of changes in the heart of the horse are far greater than in man.

We may firstly determine, in an examination of the heart, the nature of the impulse, and secondly the character of the sounds heard on listening over the cardiac area.

1. **CARDIAC IMPULSE.**—The impulse of the heart may be felt by placing the hand directly upon the left side of the chest, immediately below the elbow. It is more distinct in flat-sided and thin animals, and less so, or even absent, in round-chested and fat ones. The impulse may be altered in position and area, in force, in character, and in rhythm.

(A.) *In position and area.*—It may, owing to abdominal distension, be displaced forwards and upwards, or it may, owing to collection of fluid in the pericardium or chest, be displaced upwards and laterally.

The area over which the impulse is felt is 'extended' in cardiac hypertrophy, and in adhesions of the pericardium to the chest-walls.

It is very feeble, though extended, in pericardial effusion.

(B.) *In force and character.*—The impulse is more forcible, and sometimes extended over a larger area, in most cases of hypertrophy, with dilatation and incompetence of the valves. In these cases it is rather slow and steady, and occasionally appears to be produced not only when the heart contracts, but when it commences to dilate. This latter is called diastolic impulse.

Diminished impulse indicates feebleness of the heart. This may be due to degeneration, or to weakness of the heart owing to thinning of the walls or to general disease of the system.

It is also diminished in effusion into the sac of the pericardium, and may be lessened owing to pericardial adhesions.

(C.) *In rhythm.*—May be irregular in force, at one time strong, at another weak, or it may be irregular in time or in character.

2. **CARDIAC SOUNDS IN HEALTH AND DISEASE.**—When the ear is placed over the cardiac area, two sounds

are audible in health. They may be best heard by extension of the left fore-leg, and by pulling forward the humerus as far as possible.

They differ in character, and have been compared to the two syllables 'lab' and 'dup,' the first being longer and heavier (lab), the second, which follows quickly after a short interval, being shorter and sharper (dup). After the second sound follows another longer interval.

The first sound is complex, and although in part due to the closure of the auriculo-ventricular valves, other factors appear to be necessary for its normal production, such as a healthy condition of the muscular tissue of the heart and of the blood. It is heard during the first part of the contraction or systole of the heart.

The second sound is chiefly due to the closure of the semilunar valves in the aorta and pulmonary arteries, and corresponds in time to the first part of the diastole, or period of dilatation.

In investigating the heart's action in disease, we have to consider firstly the two sounds, and secondly the two intervals by which the sounds are separated. In health there is no sound audible during the intervals.

The natural sounds may be modified in disease, and fresh sounds may be heard. These fresh sounds are termed **murmurs**, and although they may replace the normal sounds, they always do something more, *i.e.*, *they always occupy part of the interval as well.* The natural sounds may be altered in intensity, in duration, in rhythm, in pitch (high-pitched and clear, or low-pitched and muffled), and finally in some cases they are absent altogether.

Murmurs may be heard when both sounds are present, or when one or both of the sounds is absent. The terms used to describe murmurs are *soft*, *blowing*, *loud*, *faint*, *harsh*, *grating* and *rasping.*

Cardiac murmurs are divided into two groups, endocardial and exocardial.

(A.) Endocardial Murmurs.—Murmurs in the heart are called organic when they depend upon some structural change in the heart, and inorganic when no such change exists. As the heart is continually contracting and dilating, they fall further into two groups, *systolic* and *diastolic* murmurs.

Murmurs in the heart or vessels are due to eddies set up in the blood; they are heard when a current of fluid passes through a narrow orifice into a larger space. In the heart, so long as the normal relation exists between the size of the orifices and that of the cavities of the ventricles, murmurs are not produced; but when the relation is disturbed murmurs are likely to arise.

They may depend upon dilation of the cavities into which the blood is passing, the orifices being unaltered, or to changes in the condition of the orifices, the cavities remaining natural. These conditions are usually associated. A murmur heard during the systole of the heart, called a *ventricular systolic murmur*, may be due to abnormalities in the auriculo-ventricular valves, or in the arterial orifices. When occurring at the auriculo-ventricular orifice, the murmur is owing to the imperfect closure of the valves during the contraction of the heart, so that some of the blood, instead of passing through the arterial orifices, passes back, or regurgitates into the auricles. In these cases it is a *murmur of regurgitation.*

When occurring at the orifice of the aorta, or pulmonary artery, the systolic murmur is due to thickening or other changes in the semilunar valves causing constriction of the orifice, and thus impeding the onflow of blood.

In these cases it is a *murmur of constriction.*

In the horse the systolic murmur is generally very loud

and high-pitched, especially when associated with hypertrophy of the ventricles. It has been heard in mitral insufficiency consequent upon dilatation of the left ventricle, and also where the mitral valves are thickened or otherwise changed, and where large collections of fibrinous clots have been found among the cordæ tendinea. In disease of the tricuspid valve it has also been detected.

Murmurs heard during the diastolic period, known as *ventricular diastolic murmurs*, are likewise due either to disease of the auriculo-ventricular valves or of the arterial orifices.

When occurring at the auriculo-ventricular valves the murmur is due to the passing of the blood from the auricle into the ventricle through a constricted orifice, due to disease of the tricuspid or mitral valves. It is a *murmur of constriction.*

When occurring at the arterial orifices it is due to *regurgitation* of the blood from the aorta or pulmonary artery into the ventricles, owing to the insufficient closure of the semilunar valves.

Murmurs may be heard both during diastole and systole, in which case they are said to be double.

The usual pathological changes causing the double murmur in the horse are aortic insufficiency and mitral constriction.

(B.) Exocardial Murmurs.—The healthy pericardium is smooth, and thus no sound is produced when the opposed surfaces of the serous membrane come in contact.

When, however, the pericardium is roughened, as it is when inflamed, a sound is produced. This is pericardial friction.

It is rough, rubbing, or grating in character, and being synchronous with the heart's action, is nearly always double, though it may be heard during systole or diastole

only. It appears superficial, and as though close under the ear.

When much effusion into the sac follows the dry stages of inflammation, the sound is no longer produced, owing to the separation of the roughened surfaces by the fluid.

When the pleura over the pericardium is inflamed, the movement of the heart may cause a friction by the rubbing of the two layers of the pleura. This is pleuro-pericardial friction, its origin being in the pleura over the pericardium.

GENERAL OBJECTIVE SYMPTOMS OF CARDIAC DISEASE.

Palpitation. — Palpitation, or tumultuous rhythmical action of the heart, is a common symptom in functional and organic disease of the heart or pericardium.

It occurs when there is more or less sudden demand of work from the heart in excess of its powers, as in acute or chronic diseases of the heart and pericardium, anæmia and debility, in altered conditions of the quantity and quality of the blood, as in anæmia and plethora, and in some specific and other general diseases.

If the additional strain put upon the heart be sudden and temporary, the result is only a transient attack of palpitation. If persistent, hypertrophy may be induced.

It may also be caused by nervous influences. The latter may be of reflex origin, as when palpitation accompanies acute or chronic intestinal disease ; or it may be of central or intrinsic origin.

Palpitation may be continuous, or may occur at intervals only. It is generally accompanied by increased frequency and apparent force of the heart's action, and may be regular or irregular in force and in rapidity.

Pain is rare, but there is usually anxiety and hurried breathing.

In paroxysmal attacks depending upon nerve-irritation and disturbance, there may be fainting and actual syncope.

Treatment of Palpitation.—When palpitation is dependent upon anæmia and general debility, tonics, especially salts of iron, are indicated, and the diet should be nutritious and digestible.

When it results from nervous excitement, quietude should be enjoined. Internally, digitalis and potassium, bromide and belladonna, are valuable remedies. Chloric ether, opium, and hydrocyanic acid are also of great efficacy.

In very bad cases, Fleming's tincture of aconite, in 6 to 8 minim doses, has proved very successful in the treatment of this complaint.

When the palpitation is the result of cardiac or pulmonary disease, it is treated in connection with the disease with which it is associated.

Irregularity.—Irregularity of the heart's action may affect either the rhythm or the force of the beat, and is of more serious importance than palpitation. It is probably closely related to defective nutrition of the substance of the heart itself.

It is common in dilatation of the heart.

Irregularity of force is common in the horse in incompetence of the mitral valves with dilatation of the left ventricle.

Intermittence.—Intermittence of the heart's action is a still more serious indication of cardiac failure than palpitation and irregularity. It is due to temporary arrest of the ventricular systole. It may arise from imperfect filling of the ventricle, as in fatty heart, or from great obstruction to the outflow of the blood, as in aortic obstruction. It may be of purely nervous origin.

Cyanosis is a condition very rarely met with. It only affects very young animals, and they only live a very short time. It is due to patency of the foramen ovale, and consequent mingling of arterial and serous blood.

It is manifested by blueness of the visible mucous membranes, difficulty of breathing, and coldness of the surface.

ACUTE AFFECTIONS OF THE HEART.

ACUTE ENDOCARDITIS.

Definition.—Is inflammation of the endocardium lining the cavities and valves of the heart.

Etiology.—Acute endocarditis in horses is usually associated with pericarditis, and endo-pericarditis is the most frequently met of all acute diseases of the heart. It is especially observed in rheumatism, several specific fevers, in pyæmia, and many forms of septic poisoning.

Acute endocarditis as a primary affection is probably very rare in the horse. Mr. C. Gresswell, of Nottingham, has, however, had several cases of this disease in young thoroughbred foals.

Symptoms.—Inflammation of the endocardium is in most instances attended with pyrexia, increased frequency of the pulse, and dyspnœa. When it follows in the course of rheumatic inflammation there is exacerbation of the febrile manifestations and obvious cardiac disturbance.

The heart's action is tumultuous, and apparently forcible, but the pulse is feeble. In listening to the chest we may hear pericardial friction, if the outer serous membrane is inflamed, and cooing murmurs, though sometimes audible in endocardial inflammation, are so frequently disguised by other abnormal sounds, as not to be relied upon in forming our diagnosis.

Clonic spasms of the superficial muscles of the anterior part of the body are present in many cases of endocarditis.

Owing to the uncertain character of the symptoms, endocarditis is not easily diagnosed from pericarditis.

Sequelæ.—Inflammation of the endocardium is usually followed by structural changes of the valves, and this change induces dilatation and alteration in the cavities of the heart. Weakened cardiac action and dropsical effusions may follow in various parts of the body.

Morbid Anatomy —The endocardium consists of a layer of connective-tissue with an endothelial lining. The inflammation is more marked round the orifices and in connection with the valves and their appendages, and, in rheumatism, particularly in the valves. During the inflammatory process the deeper layers of the endothelium become rapidly infiltrated with young cells, and the inter-cellular substance becomes softened and destroyed, and thus there is formed a soft tissue composed entirely of cells. This new tissue projects through the endothelium in the form of minute granulations and vegetations upon the surface of the softened valve. There is also more or less increase of vascularity in the external endothelial layers; but the above changes occur in an almost non-vascular tissue.

The roughened granulations act as foreign bodies, and thus frequently induce coagulation or deposition of fibrin upon themselves.

The cell infiltration may break down, and form an endothelial ulcer or a small abscess. The inflammatory process is generally, however, less intense and malignant. The granular valves may then adhere to each other or to an inflamed patch in the wall. The new cell growths become organized into a fibrinated structure, whilst in part it undergoes fatty and calcareous degeneration.

These changes produce permanent thickening, rigidity, and shrinking of the valves, and consequent insufficiency or stenosis, or both.

The new tissue may continue to grow after the process has subsided, and thus are produced vegetation and papillary excrescences on the valve. These consist of a lowly organized tissue, which tends to undergo fatty and calcareous changes.

Treatment.—The treatment resolves itself chiefly into the treatment of the diseases with which the endocarditis is associated.

Cardiac sedatives are indicated when the irritability and pain are great, but must be given with care. Bleeding is recommended by some; but as it probably only gives temporary relief, and probably favours coagulation and the deposition of the fibrin on the roughened surfaces, it is best not to adopt this measure.

In cases where debility and exhaustion follow, stimulants in moderate doses are necessary.

MYOCARDITIS.—The muscular substance of the heart may be the seat of inflammation, whereby it becomes softened or, more rarely, indurated. Intense and concentrated inflammations, leading to the formation of abscess, may occur as the result of a pyæmic process. Myocarditis rarely occurs except in connection with disease of the endocardium and pericardium, whence the inflammation spreads to the muscular tissue itself. It is an occasional sequel of various acute fevers, and of pyæmia and other septic conditions of the blood.

It gives no distinctive symptoms during life by which it may be recognised, but the heart's action is weak and irregular.

CHRONIC AFFECTIONS OF THE HEART.

ENLARGEMENT OF THE HEART.—Hypertrophy and Dilatation.—Enlargement of the heart may result from increase in size of the muscular walls, or from increase in the capacity of the cavities. When the heart is hypertrophied without any alteration in the capacity of the cavities, the condition is termed *simple hypertrophy.* When there is increase of capacity of the cavities, without enlargement or multiplication of the muscular elements or thinning of the walls, it is termed *simple dilatation.* When the muscular structure is increased, and there is also increase in the capacity of the cavities, it is termed 'hypertrophy with dilatation,' or *eccentric hypertrophy.* It is doubtful if hypertrophy with diminution of cardiac capacity occurs, but it has been mentioned under the term *concentric hypertrophy.*

Etiology.—Hypertrophy with or without dilatation may, in most instances, be regarded as a compensatory condition, owing to some obstruction to the circulation. It may be induced by long-continued overwork; and in hunters and racers hypertrophy, or hypertrophy with dilatation, is of frequent occurrence. Hypertrophy may also result from frequently recurring or persistent palpitation.

The obstruction to the circulation may occur at the cardiac orifices, or at the orifices of the large arteries, especially at the mitral and aortic openings. The obstruction to the blood-flow may be in the pulmonary circuit, as in bronchitis and emphysema, and occasionally it may be in other organs. Dilatation with hypertrophy is a frequent result of aortic or mitral incompetence, owing to the regurgitation of the blood.

Symptoms.—Vary widely, according to the seat and extent of the enlargement, and to the diseases with which it is associated.

In many cases, where the hypertrophy is compensatory, there are no symptoms. When the hypertrophy is considerable and in excess of the dilatation, the arteries tend to become fuller, the veins less full, and the circulation is accelerated. The pulse, at first full, becomes feeble as the dilatation increases.

The impulse of the heart is increased and is sometimes prolonged; and in very marked hypertrophy is often observable at a distance from the animal, and may be distinctly felt.

The heart-sounds may be more intense, but are usually less distinct.

When dilatation is in excess, the veins tend to become over full, the arteries less full, and the circulation retarded. The pulsations are smaller, feebler, and more irregular or intermittent; and there may be dyspnœa, especially when exertion is made. In advanced stages there is coldness of the extremities, and a tendency to œdema or dropsical effusions. The jugular venous pulse is most often noticed when the right side is dilated. So long as the hypertrophy keeps pace with dilatation there may be no symptoms; but when the hypertrophy ceases to progress, while the dilatation increases, symptoms are soon manifested from impairment of the heart's action. Exertion in this case is attended with much difficulty.

Morbid Anatomy.—The heart is increased in weight and bulk, the relative extent of which depends upon the relation between the hypertrophy and the dilatation. The shape is also often modified according to the parts involved, and is generally rounder than natural. The auricles are much less frequently enlarged than the ventricles, and the left ventricle more often than the right.

ATROPHY OF THE HEART.—Atrophy, or decrease of substance of the heart from diminution in the size or in the

number of the muscular elements, is general or partial. It may be attended by thinning of the walls and dilatation of the cavities, when it is called *eccentric atrophy;* or it may occur without alteration in the capacity of the cavities, when it is called *simple atrophy.* There is much weakness with feeble cardiac impulse, which is felt only over a small area; but there are no diagnostic symptoms of this condition.

CHRONIC VALVULAR AFFECTIONS. — Valvular disease may arise from acute or chronic endocarditis; from atheromatous or calcareous changes; from enlargement of the orifices; and, lastly, from congenital malformations.

It has been already mentioned that disease of the valves may allow of regurgitation of blood owing to insufficient closure; or may cause obstruction to the onflow of the blood, owing to constriction or partial blocking of the orifice, resulting from adhesion of the valves or thickening of the structures around the opening.

Each orifice is then capable of each kind of disturbance: regurgitation and constriction. There may be theoretically, therefore, eight kinds of murmurs, viz.: (1) Aortic regurgitation and constriction; (2) Mitral regurgitation and constriction; (3) Pulmonary regurgitation and constriction; (4) Tricuspid regurgitation and constriction.

Auscultation is attended with great difficulty in the horse, and, in consequence, our means of diagnosing valvular affections are far more limited than in man.

Disease of the left side is more common than on the right. In disease of the valves of the left side, the pulse is altered in character and rhythm; while in disease of the right side, the chief manifestations are in the venous circulation. Jugular pulsations are very commonly visible, and dropsical effusions and anasarcous swellings liable to follow. Yet it

is to be remembered that, in disease of the left side, the right side may soon become disordered in consequence.

In the horse, the mitral and aortic valves are more specially liable to be diseased. In many instances both valves are involved simultaneously. Probably the commonest form of valve disease met with in the horse is aortic obstruction and mitral regurgitation. During auscultation, if we are in doubt whether a murmur occurs during the systole or diastole, we may feel the pulse at the jaw or fore-arm while we listen. If the murmur is *systolic*, it will of course be heard at the same time, or nearly so, *as the pulse beat is felt;* if *diastolic*, it will be heard *between the beats of the pulse.*

Mitral Regurgitation.—At each systole of the left ventricle blood flows back or regurgitates into the left auricle. The pulmonary circulation thus becomes overloaded, and is subjected to increased pressure. This difficulty is met, for a time, by compensating hypertrophy of the right ventricle. Hence, during this stage there are no symptoms of ill-health, though there is not unfrequently difficulty of breathing during exertion. After a time the hypertrophy of the ventricle gives way to dilatation. Thus the engorgement and tension of the pulmonary vessels is increased and reflected back through the right side of the heart to the systemic veins, which now also become overloaded, just as they do from the first in cases of disease of the valve of the right side. In consequence of the mitral regurgitation being no longer compensated for by increased force of the right ventricle, the aorta now becomes under-filled.

Hence symptoms of systemic venous congestion follow. Pulmonary complications with cough are sometimes present, the breathing is disturbed, and there is severe dyspnœa when the horse is much excited or during severe exertion.

The pulse is small, feeble, and often irregular and intermittent.

Mitral Obstruction.—The clinical history of mitral obstruction is similar to that of mitral regurgitation, with which it is in many cases associated. In the horse we do not know that the murmur characteristic of this disease in man has ever been distinguished. This murmur in man immediately precedes the systole, and is therefore termed presystolic. It is due to the passage of the blood from the left auricle through a constricted mitral orifice.

Tricuspid Regurgitation.—In this disease, at each contraction of the right ventricle blood passes back through the tricuspid valves into the right auricle, and thence into the venæ cavæ and jugulars, which thus become rapidly dilated and overloaded.

A reflux pulsation in the jugulars with each ventricular contraction is pathognomonic of tricuspid regurgitation.

In this disease a systolic murmur is heard, as in mitral regurgitation, but we cannot distinguish the two murmurs in the horse.

Aortic Obstruction.—When the aortic semilunar valves obstruct the onflow of blood through the orifice, a murmur is produced *during systole.* When they allow of regurgitation back into the ventricle, the murmur is *diastolic.*

When both conditions are present, both murmurs are produced. In obstructive disease, the left ventricle hypertrophies to overcome the resistance; but after a while the mitral orifice, with the valves there, becomes involved, and the equilibrium breaks down. In consequence of this, the symptoms of mitral regurgitation are manifested.

Aortic Regurgitation.—In this disease the blood-flow is retarded; for after every contraction of the left ventricle, part of the blood regurgitates back from the aorta into the ventricle. The left ventricle therefore hypertrophies,

in consequence of the increase of work thus thrown upon it.

After a time the hypertrophy gives way to dilatation, owing to impaired nutrition and degeneration of the cardiac walls, arising from imperfect filling of the coronary arteries during the ventricular systole.

Implication of the mitral valve follows after a while, and the symptoms of mitral regurgitation are manifested.

The pulse in aortic regurgitation is sudden and jerky, and is termed the 'water-hammer pulse,' or 'Corrigan's pulse.'

In aortic obstruction the pulse is not materially altered.

TREATMENT OF CHRONIC VALVULAR DISEASES.—The treatment of the valvular diseases can only be palliative, and in most instances it is not an object to prolong life in horses incapable of exertion. Regulation of work, which should not be in excess of the powers of the animal, and good diet, are the first essentials in the treatment of cardiac diseases.

Tonics, such as iron, nux vomica, and other vegetable tonics, are indicated in most cases, but we must, in all cases, treat the symptoms as they arise. When there is great irritability of the heart, belladonna, bromide of potassium, and chloric ether are of service in alleviating the distressing symptoms. Digitalis is of great efficacy as a heart tonic, and is indicated whenever cardiac failure is threatening.

DEGENERATIVE CHANGES OF THE HEART.

FATTY DEGENERATION OF THE HEART.—Under the term fatty degeneration are included two different pathological conditions. These are fatty metamorphosis, or degeneration proper, in which the fat is derived from the metamorphosis of the cardiac muscle, and fatty degeneration, where it is derived from the oleaginous, saccharine, or nitrogenous principles of the food.

These two conditions may occur together, and are not unfrequently associated with other abnormal conditions of the heart, as partial or general dilatation.

Fatty Infiltration.—In health there is a certain quantity of fat covering the surface of the heart, beneath the visceral layer of the pericardium. Under certain circumstances—for example, from want of sufficient exercise—the fat tends to accumulate around the base and origin of the great vessels, and also to gradually insinuate itself between the muscle fibres. The fat is always more abundant near the surface.

Fatty Metamorphosis.—This condition may affect the cardiac muscle throughout, or in portions only. The muscle becomes softer and more friable, and often breaks with a granular fracture. It is more flabby, paler, and more opaque than normal cardiac muscle. True fatty degeneration is mostly met with in cases of dilatation of the cavities with thinning of the walls of the heart. It is also a not uncommon result of aortic incompetence, in which disease the circulation in the coronary arteries is interfered with, and the heart muscle is badly nourished in consequence.

Diffuse fatty degeneration sometimes occurs in acute specific fevers.

There are no diagnostic symptoms of fatty change in the heart, though degeneration renders the organ weak and incapable of performing any unusual exertion, or, if advanced, of even carrying on the normal circulation.

CALCAREOUS DEGENERATION OF THE HEART.—Cases of calcareous change in the cardiac muscle of the horse are very rarely met with. Only two cases are recorded, and in each the right auricle only was calcified.

RUPTURE OF THE HEART.—Rupture of the heart is very rarely met with. In most instances it is due to some previous pathological condition of the organ. The imme-

diate causes are over-exertion and shock, resulting from a fall or from direct violence to the chest.

It is of most frequent occurrence in aged horses, after sharp exertion.

The usual seat of the rupture is at the conus arteriosus of the left ventricle, where the tendinous ring unites the great aorta with the ventricle.

Mr. Percival records an instance of rupture of the right auricle in the horse occurring shortly after a race.

NEW FORMATIONS IN CONNECTION WITH THE HEART.—The new formations met with most commonly in the heart of the horse are malignant and parasitic growths, cardiac polypi, and vascular tumours.

The effects of these growths on the health of the animal vary chiefly in accordance with their situation, and also, but to a less extent, with their nature.

Occasionally the heart is the seat of malignant growths, which though possibly originating in the organ itself, are with greater probability regarded as sequels of the general invasion of the system by cancerous growths.

Hydatid cysts have been described in connection with the heart in cattle and sheep, and they may also occur in the horse.

Single individuals of strongylus armatus have been found in the pericardial sac of young horses by Professor Robertson.

Vascular tumours in the heart are due to dilatation of the veins and capillaries beneath the endocardium.

Of all cardiac growths, the most frequently met with are the so-called fibrinous polypi, which seem to originate from the muscular tissues of the heart. They are composed of laminated fibrin, the central parts being more distinctly organized and fibrillated.

Cardiac tumours cannot be diagnosed during life.

ANGINA PECTORIS.—In man, angina pectoris (ἀγχω, to strangle ; pectus, the breast), or breast pang, is a disease of the heart characterized by paroxysmal attacks of neuralgic pain in the chest, and is rarely met with under 45 years of age. This affection is believed by Williams to affect the horse sometimes, and, indeed, from the frequent occurrence of heart disease in this animal, this is only what we might expect. This author describes a case in an aged horse brought to him for examination. For twelve months the animal had done very little work, owing to the fact that when excited by work he manifested severe agony in the near fore-limb. In this case the heart was manifestly diseased.

DISEASES OF THE PERICARDIUM.

ACUTE PERICARDITIS.

Definition.—Inflammation of an acute type affecting the pericardial membrane.

Etiology.—Acute pericarditis may appear as a primary or idiopathic affection from cold, damp, exposure or fatigue. It is, however, most frequently met with in association with other diseases, rheumatic fever especially. It is also often associated with strangles, influenza, purpura, pyæmia, and other septic conditions of the blood. It may result from spreading of inflammation from the lungs and pleura, or from the irritation of new growths, or from parasites in the sac.

Finally, it may be induced by wounds or by fractured ribs.

Symptoms. — The symptoms of pericarditis are very variable, and when unassociated with general disease may be very indistinct and obscure. Generally pericarditis is met with in association with endocarditis, and in these cases the symptoms are very difficult to distinguish.

The disease, when severe, is usually ushered in with considerable fever. The pulse is short, hard, irritable, and accelerated, and the heart's action is often irregular. Sometimes the pulse is bounding and violent; sometimes, when there is much effusion, it is feeble and fluttering. The respirations are accelerated; the extremities are cold; cough is often present, and there is pain, especially during movement. In less severe cases there is less constitutional disturbance, and correspondingly less pathological change.

Debility is sometimes very marked.

Associated with the pericarditis, spasms of the superficial muscles have often been observed. These spasms or cramps are in most instances confined to the muscles of the pectoral region and neck, but occasionally, also, the posterior extremities are affected.

The friction characteristic of pericarditis in man is not always detected in the horse. When heard, it is a rasping, grating to-and-fro sound, synchronous with the cardiac action.

If this sound be associated with a deeper seated murmur in the heart, endocarditis is also present. When the effusion supervenes the friction disappears, and is replaced by a dull churning noise of the heart movements. If the effusion be considerable, the pulse becomes feeble, fluttering, and irregular. There may also be visible carotid and jugular pulsation, with great distress in the breathing, and œdema may appear in various parts.

In influenza, pericarditis is a somewhat frequent complication.

The symptoms in this form of pericardial inflammation vary much, being sometimes well marked and sometimes very indistinct. They are best marked in cases where the lungs and pleura are free from disease.

Prognosis.—Pericarditis may terminate in partial recovery

with adhesions, in complete recovery, or in death (usually by syncope). The prognosis is not unfavourable except in very severe cases.

Morbid Anatomy.—The anatomical changes in pericarditis vary considerably with the severity and nature of the attack. On the whole they resemble those already described in inflammation of the pleura.

The effusion, which is sero-fibrinous, is usually considerable, and when small may soon be absorbed.

Complete restoration is rare, for more or less extensive adhesions and agglutination usually persist. In many cases the plastic material effused spreads over the surface of the heart, assuming a reticulated appearance of considerable thickness.

In some cases the effusion is blood-stained, and in others it may mingle with pus. In the pericarditis of acute rheumatism and epizootic fevers the cardiac muscle and the endocardium are also generally involved.

Treatment.—In the early stages of primary pericarditis, with a full hard pulse, Fleming's tincture of aconite, with nitric ether and liquor ammoniæ acetatis is recommended in moderate doses. If the pain continue unallayed, tincture of opium should be substituted for the aconite. Digitalis does not seem to have a beneficial action in pericarditis. In the early stages some recommend moderate bleeding, which they repeat a second time if necessary, or follow up the first abstraction of blood by the administration of Fleming's tincture. Under no circumstances should bleeding be adopted, except to relieve urgent symptoms in the early stages, as in pericarditis there is great tendency to early diminution of the cardiac power.

Woollen cloths wrung out from hot water should be wrapped round the chest, as in pleurisy, and should not be re-applied for a couple of hours. During the intervals the

left side may be well rubbed with some stimulating liniment containing tincture of opium. The legs should be carefully bandaged, and the food should be nutritious and easily digestible. After the early stages are over and effusion sets in, a cantharides blister applied over the cardiac region is very useful. Stimulants and tonics, such as the salts of iron, with diuretics or potassium iodide, may now be given internally.

When the pericarditis is associated with rheumatism, the treatment does not materially differ from that above described. Colchicum is useful internally, but aconite or opium are perhaps preferable. Bicarbonate of potash and nitric ether may be given in addition; and the woollen cloths should be applied to the chest as in the idiopathic variety.

When associated with the specific fevers, pericarditis should not be treated apart from the general disease. Moderate blisters may be applied to the left side, and stimulants and potassium iodide given internally. When the fluid in the pericardium remains unabsorbed, and there is in consequence serious interference with the heart's action and great difficulty in breathing, the fluid should be removed by paracentesis or puncturing of the pericardial sac.

HYDROPS PERICARDII.—Dropsy of the pericardium, or hydrops pericardii as it is termed, may follow an attack of subacute pericarditis, but in most instances it is part of a general dropsy.

The fluid is purely serous, thus differing from that of acute pericarditis. There are usually no symptoms by which this condition can be diagnosed during life.

CHAPTER VII.

DISEASES OF THE ALIMENTARY SYSTEM.

GENERAL REMARKS ON DISEASE OF THE ALIMENTARY CANAL.

Diseases of the alimentary canal are in most instances due to dietetic errors.

We have already considered several diseases, diabetes, broken wind, oxaluria, azoturia, and others, which are also traceable to errors in feeding. Diseases of the alimentary tract are especially important from two points of view, for not only are they among the most common of all forms of diseases in the horse, but they are at the same time the most easily guarded against by careful management of the diet.

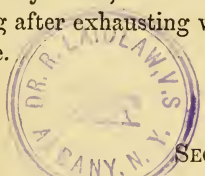
In the horse the intestinal tract is more liable to disease than the stomach, whereas in the ox and sheep the stomach more frequently suffers. This is in all probability due to the fact that in the horse the stomach is much less complex than in ruminants, and is also smaller in proportion to the rest of the intestines than in the cud-chewing animals.

In consequence of this the process of digestion begun in the stomach is largely completed in the intestines in the horse.

Professor Williams points out the important fact that easily digested food taken in excess is liable to derange the

small intestines, whereas coarser and more indigestible food containing much woody fibre, as over-ripe hay, rye-grass and coarse straw, is more apt to accumulate in the large intestine, causing disordered action, inflammation, or even paralysis of the intestinal muscular tissue. The same authority also mentions that boiled food is apt to be retained in the stomach, and if given in excess may cause distension, inflammation, paralysis, and even rupture.

Lastly, it should be remembered that it is not only the quality of the food which may set up disorders in the alimentary tract, but that irregularity in diet and full feeding after exhausting work are also very liable to induce disease.



SECTION I.

DISEASES OF THE MOUTH AND THROAT.

STOMATITIS.—Inflammation of the mouth occurs in several forms, and we have already considered a contagious disease of the horse in which the specific pustular formations have their special seat on the buccal membrane.

Causes.—Stomatitis is most frequently met with in young animals, and is especially predisposed to by malhygienic conditions and improper dieting. It may be due to local irritation or to mechanical causes, and is sometimes produced by extension of morbid action in the parts near, or may follow disorders of the alimentary canal.

It often occurs in animals debilitated by disease, and is in many instances traceable to certain vegetable fungi, or to a special acarus. The oidium albicans, one of the hyphomycetes, has long been recognised as the active cause of some forms of stomatitis.

Varieties and Symptoms :—

Simple, or Catarrhal Stomatitis.—This form is chiefly met with in foals. It first appears as small circumscribed red patches on the buccal membrane of the cheeks and roof of the mouth. These patches are covered with a yellowish film, which soon separates, and leaves a superficial erosion.

The excoriated patches gradually coalesce, and thick saliva, mingled with the products of inflammatory action, accumulates in the mouth. There is an unhealthy fœtid odour in the mouth, and the buccal membrane is somewhat thickened and congested.

This form is occasionally seen in old animals, and in many instances is associated with disordered digestion.

Vesicular Stomatitis affects both young and old animals.

It may accompany the simple variety, but generally occurs as an independent affection.

In this form small vesicles appear on the buccal membrane of the cheeks, around the angles of the mouth, and on the sides and frænum of the tongue.

They soon rupture, and leave minute ulcers, several of which frequently coalesce.

Pustular Stomatitis.—The vesicles in the preceding form may develop into pustules, and thus this variety is sometimes a sequel of vesicular stomatitis. In most instances, however, pustular stomatitis first appears as small yellowish spots, developing into distinct pustules, which rupture, and leave small, deep ulcers.

Treatment of Stomatitis.—The hygienic and dietetic arrangements should be attended to in the first place.

As local applications, chlorate of potassium, 15 grains to the ounce, or carbolic acid, 1 part in 40, are recommended. If the ulcers become indolent, they may be touched with a solution of sulphate of copper, a scruple to the ounce, or silver nitrate, 10 grains to the ounce.

Internally, antacids, such as bicarbonate of potash, with vegetable tonics are required in some cases, and they may be preceded by the administration of a mild purgative.

GLOSSITIS.—Inflammation of the structure of the tongue is rarely met with in the horse, except when resulting from the action of irritants, or from mechanical injury. It may follow on inflammation of the neighbouring structures.

The tongue becomes swollen, tense, and painful from the inflammatory infiltration, and soon protrudes in consequence of its increase in size. There is difficulty in swallowing, and ropy saliva and mucus accumulate in the mouth.

Treatment.—Gargling, firstly with warm water, and afterwards with antiseptic or astringent lotions, is recommended. Laxatives, if required, must be given per rectum.

PAROTITIS.—Inflammation of the parotid gland may affect one or both sides, and is a frequent accompaniment of strangles.

Parotitis is acute or subacute, and is often met with associated with or supervening on various febrile attacks.

Symptoms.—When of an acute type, parotitis may quickly terminate in abscess of the gland. The inflammation, however, is usually of slower progress, and reveals itself by gradual swelling and tenderness of the gland, with difficulty in moving the head and protrusion of the nose. The inflammation may gradually subside, or it may terminate in abscess, which, if not opened, may burst into the pharyngeal pouch.

When the inflammation is very acute, the whole gland may be affected; but when not so intense, it is usually limited to a portion of the gland-structure.

Treatment.—Hot fomentations or poultices may be applied for a time, and if pus forms it should be liberated.

If the inflammation proceeds slowly, a cantharides blister or tincture of iodine of three times the B.P. strength may

be applied. Internally, tonics and stimulants are indicated.

PTYALISM—SALIVATION.—Ptyalism or salivation is an abnormal increase in the amount of secretion of saliva, which dribbles away continuously, or collects in foam around the mouth.

We do not apply the term salivation or ptyalism to the escape of saliva from the mouth resulting from inability to swallow it.

The secretion of saliva is a reflex process, and salivation depends therefore either upon peripheral irritation, or upon undue action of the salivary nervous centres, or on both these conditions.

It is generally dependent on peripheral irritation occurring in some diseases of the mouth, pharynx, throat, and other organs; and may also result from irritation caused by irregularity of the teeth, or foreign bodies in the mouth. It may occur in rabies and tetanus, and in some instances no assignable cause is found.

Finally, it may be due to the administration of certain drugs.

Treatment.—In all cases the cause should be ascertained and removed, if possible.

If there be any irregularity of the teeth, this should be attended to.

Gargles of alum or chlorate of potash are often very useful in these cases. If the salivation continues, belladonna is worthy of trial.

ACUTE PHARYNGEAL CATARRH.—Inflammation of the mucous membrane and submucous tissue of the fauces and pharynx are common in influenza, strangles, and other specific fevers in which the air-passages are involved.

This form of pharyngitis we have already alluded to, as also we have to inflammation of these parts occurring as

part of general catarrh. But this disease also occurs as an independent affection, from cold, exposure, damp, direct irritation, or disordered digestion.

Symptoms.—Deglutition is attended with difficulty, and sometimes with coughing, during which the food is expelled back into the mouth, and liquid through the nostrils. There is no fever or impairment of respiration unless laryngitis is also present.

Treatment.—Many cases recover in a few days with rest. Inhalation of hot-water vapour medicated with carbolic acid or opium is very useful.

Gargling with solution of chlorate of potash or sulphurous acid may also be tried, and hot fomentations externally are useful in allaying the irritation.

In chronic cases, stimulating liniments or blisters may be applied.

POST-PHARYNGEAL ABSCESS.—Simple inflammation of the pharynx may terminate in the formation of abscess in the pharyngeal structures.

Suppuration in the parts, however, is more frequently met with in the pharyngitis occurring in some of the specific fevers, or in animals which have been exposed to malhygienic conditions. Abscess may also occur here as part of a general pyæmia.

Suppuration in the pharynx causes obstruction to deglutition, the breathing becomes difficult, and the throat is generally tender.

Treatment.—The treatment of pharyngeal abscess is essentially surgical. If the breathing become difficult, inhalation of hot-water vapour, and the hot fomentations externally, are useful. If the abscess do not burst, it may be necessary to open it, especially if breathing is very much interfered with.

Section II.

DISEASES OF THE ŒSOPHAGUS.

INFLAMMATION OF THE ŒSOPHAGUS.—Inflammation of the œsophagus is generally the result of direct injury. It may be induced by the passage of very large or hard portions of food, or by the administration of irritant liquids. It may be caused by external violence, and not unfrequently occurs as part of general catarrh of other mucous membranes.

Symptoms.—Difficulty of swallowing; tenderness on manipulation, and sometimes spasm of the œsophagus, are the usual symptoms manifested. In severe cases there may be febrile disturbance. When the inflammation occurs as the result of the administration of irritant liquids, as, for example, stimulating liniments given inadvertently, too hot water, or some poisons, as arsenic in solution, the submucous tissue may be much infiltrated. Perforation of the coats has been noticed in cases where the inflammation has been very severe.

Treatment.—Many cases do not require treatment beyond the use of liquid food for a day or two.

In severe cases linseed gruel, to which a little tincture of opium is added, is of benefit where there is much pain. Hot-water fomentations, followed by the application of stimulating liniments, are useful externally. Stricture of the œsophagus often follows severe inflammation.

STRICTURE OF THE ŒSOPHAGUS.—Stricture of the œsophagus may be left as the result of previous inflammation, or it may depend on tumours in connection with the walls or in the neighbouring structures.

Symptoms.—Gradual progressive difficulty in swallowing. The food often accumulates above the constriction, and the œsophagus above may become dilated and hypertrophied in consequence. Ejection of the accumulated liquid often follows.

Treatment.—Nutritious diet is required, and should be semi-solid or liquid. It is best to pass a probang in order to ascertain the cause of the obstruction and its extent. The stricture may be dilated in some cases, and recovery follow, but dilatation of the tube above the constriction will most likely remain. In stricture due to changes in the walls themselves, treatment will in all probability be of little avail.

When it is the result of tumours, the treatment will depend on the site and nature of the abnormal growth.

SPASM OF THE ŒSOPHAGUS.—Spasm of the œsophagus is not common in the horse, and the causes inducing it are uncertain. In some instances, however, it has been shown to depend on local irritation.

Symptoms.—When food, especially of a solid nature, is swallowed, it is, after travelling a certain distance, arrested for a time, and afterwards passes on, or is ejected suddenly, through the nose chiefly, but also through the mouth. The neck is usually bent, and the œsophageal muscles may be observed to undergo contraction.

Treatment.—If any obstruction is suspected, it is well to pass the probang, and morphia in full doses may be injected subcutaneously.

If the general health is disturbed, laxatives followed by tonics are indicated.

Section III.

DISEASES OF THE STOMACH.

GENERAL REMARKS ON SOME GASTRIC SYMPTOMS.

Flatulence.—Flatulence, or distension of the stomach and bowels with gas, results from fermentation, in the course of which carbonic acid chiefly, and other gases, such as sul-

phuretted and carburetted hydrogen, are also occasionally set free. Flatulence of this kind is probably due to alteration in the quality of the gastric juice, to feeble movements of the stomach and intestines, and to pyloric or intestinal obstruction, which, by retaining the food and impeding the process of digestion, favour fermentative and putrefactive changes.

Vomiting, or Emesis.—The rarity of vomiting in the horse has been attributed, firstly, to its small susceptibility to the action of nauseants; and, secondly, to the supposed difficulty attending the process from the anatomical conformation of the stomach in the horse. It is well known that the mucous membrane is gathered in folds or rugæ around the cardiac opening, and thus the food is prevented from escaping into the œsophagus.

Vomiting in the horse chiefly depends on reflex irritation, due to functional or organic diseases of the stomach. The ejected matters are chiefly expelled through the nostrils.

Vomiting has been observed in cases of gastric distension, and Robertson mentions some cases of young horses in which sickness was induced by eating haws. Some of these cases proved fatal, and in others marked prostration for several days followed the vomiting, which was attended with great distress. Vomiting also occasionally results from the injection of certain poisons, administered wilfully or with good intention. Every case of hellebore poisoning which we have been called upon to treat has been attended with nausea and several with actual vomiting.

Lastly, vomiting occurs in cases of rupture of the stomach, and has been noticed when there was dilatation of the lower part of the œsophagus close to its opening into the stomach.

The treatment of emesis will vary in accordance with the disease with which it may be associated, under which it will be again referred to.

INDIGESTION OR DYSPEPSIA.

Varieties: (1) Acute, and (2) Chronic Dyspepsia (δυς, with difficulty; and πεπτω, I digest).

1. ACUTE DYSPEPSIA, OR INDIGESTION WITH ENGORGEMENT. — Etiology.—Acute indigestion results from engorgement of the stomach with food, from imperfect mastication, and from the ingestion of indigestible material, or food specially apt to undergo fermentative changes. Cooked food, brewers' grains, maize, musty hay, and ripe vetches, are especially liable to cause impaction. Wheat and barley are also very likely to induce indigestion, and they frequently also cause purgation, laminitis, and may even lead to a fatal result.

Horses are more liable to dyspepsia after severe or prolonged exertion, especially if the food be difficult of digestion, or in too large a quantity.

Symptoms.—The symptoms of acute indigestion are generally sudden in their onset. There is fulness of the abdomen, and the horse is restless and shows indications of colicky pain. He lies down and rises again alternately, and paws the ground with his fore feet. Eructations of wind, occasional discharge of saliva from the mouth, and tremors of the superficial muscles, particularly those of the left shoulder, are also among the symptoms of acute indigestion.

In severe cases the pain is very acute, and the horse throws himself about wildly, and frequently looks towards his flanks. The pulse and respiration are accelerated, and in some instances there are attempts at vomition, and the discharge of brown fluid contents of the stomach with the eructations.

In some instances there is no manifestation of gastric pain, but the horse remains dull and semi-comatose, and the breathing may become stertorous; he refuses his food, is moved

with difficulty, and attempts to press his forehead against a wall or tree, or anything which comes in his way. Under these circumstances the respiration is not much quickened, and the pulse is of full volume, but not so accelerated as when gastric pain is a prominent symptom. Such cases as these were spoken of by the older writers as *stomach staggers, and seem to be comparable with that form of gastric disturbance in man which is attended with vertigo and flatulent dyspepsia.*

Prognosis.—In mild cases the prognosis is very favourable, and many recover without any treatment. In severe cases the prognosis must be more guarded, as death sometimes ensues from rupture of the stomach owing to the great distension of this organ, or from gastritis.

Treatment.—In the treatment of acute indigestion a purgative should be given at once in order to clear the stomach. For this purpose aloes is preferable to any other aperient, though oil or saline purgatives are recommended by some. If there be much flatulence it will be well to give a full dose of the aromatic spirit of ammonia. If there be much pain, an ounce to an ounce and a half of sulphuric ether, with half an ounce to an ounce of spirits of chloroform, may be administered in water or gruel, every two or three hours, as long as the pain continues to be severe.

In ordinary cases a draught composed of sulphuric ether, spirit of chloroform, and aromatic spirit of ammonia, given three or four times daily, will be found very beneficial.

Hyposulphite or sulpho-carbolate of soda may also be mentioned as useful remedies in dispelling flatulence, and hydrocyanic acid in 20 or 30 minim doses is very valuable in subduing gastric irritation.

Fleming's tincture of aconite is recommended in moderate doses by some eminent authorities, but we cannot speak very favourably of its value in this disease. In cases where

there is any difficulty in administering liquids, acetate of morphia may be injected hypodermically.

If the bowels are not acted upon after the lapse of forty-eight hours or so, it is advisable to again administer some purgative medicine, but it is best not to repeat the aloes. Castor oil, in doses of one to two pints, will be found to answer the purpose as efficiently as anything else.

The treatment of indigestion with coma and cerebral complication does not materially differ from that above described.

Some have recommended the early abstraction of blood in these cases, but we agree with the view that this measure, if not harmful and calculated to impede recovery, is rarely of any advantage in the treatment of this disease. An aloetic purge, followed by the administration of sulphuric ether, spirit of chloroform, with full dose of potassium bicarbonate, is generally all that is required.

In all cases of acute indigestion which come before our notice it is necessary, firstly, to inquire carefully into the dietetic arrangements, and to advise the owner to have them more carefully regulated for the future.

In the treatment of this disease, as soon as the appetite returns, the diet should be at first of a laxative nature, and limited in amount.

2. CHRONIC INDIGESTION.—Etiology.—Chronic indigestion, like the acute form, is chiefly caused by dietetic errors, though these are not always apparent.

Sometimes the food, though of good quality, is too stimulating and dry; in other cases it is not sufficiently masticated, perhaps owing to irregularities in the teeth, but in most cases it will be found to be of inferior quality or administered irregularly.

Dietetic errors in their turn induce changes in the gastric juices and in the movements of the stomach, which, however, are sometimes deficient from impaired nerve-power.

Symptoms.—The symptoms of this affection are very variable. The appetite may or may not be impaired. Sometimes it is capricious and perverted. In other cases, though it continues good, the animal still continues to lose flesh. The bowels are generally irregular, the fæces often coated with mucus, and there may be great thirst and acid eructations. Abdominal pain is not unfrequent in severe cases, especially when the appetite remains unimpaired.

The horse is weak, sweats easily, and the skin is dry and hard.

Treatment.—The cause of the disorder should firstly be inquired into, and the dietetic arrangements carefully regulated.

If the disease proceed from imperfect mastication, due to dental irregularities, these should at once be attended to.

Some purgative should be given in all cases, unless the bowels are freely open, and should be followed by the administration of vegetable tonics, as gentian, nux vomica, quassia and chiretta, with full doses of potassium bicarbonate.

The diet should be limited in amount, and it will be advantageous to change it. When acidity of the stomach is a marked symptom, such antacids as magnesia, chalk, or subnitrate of bismuth may be administered. In the latter stages, nux vomica or quinine, with dilute mineral acids, may be given two or three times daily. Fowler's solution, given in the water with bicarbonate of potash, is also a useful means of restoring tone to the disordered stomach.

INFLAMMATION OF THE STOMACH—GASTRITIS.

Varieties.—(1) Acute; (2) Chronic Gastritis.

1. ACUTE GASTRITIS.—Etiology.—Though acute inflammation of the stomach is said to have been met with

in the horse as an idiopathic affection, yet in most instances it results from irritation of the gastric mucous membrane. Sometimes it is an accompaniment of intestinal disease.

Irritation of the gastric mucous membrane is chiefly owing to dietetic errors. Under these circumstances it may be due to impaction of the stomach, or be traceable to constant dietetic mismanagement.

Irritation of the mucous membrane may also be due to the presence of foreign bodies in the stomach, or to the ingestion of certain poisons. In influenza, and in some other specific fevers, gastritis may accompany or follow after the primary disease.

The poisons which most frequently cause inflammation of the stomach are arsenic, mercury, antimony, copper, and sometimes lead salts. The foliage of some trees, as the yew, may also cause gastritis.

Symptoms.—When gastritis accompanies impaction of the stomach, there are no diagnostic symptoms by which these conditions may be distinguished. In most cases great abdominal pain, acceleration of respiration, and a quick, rather hard, wiry pulse are among the chief symptoms of acute gastritis.

For the special symptoms of the different poisons we must refer the reader to the chapter on toxicology.

Morbid Anatomy.—The changes in the mucous membrane of the stomach vary in character and extent with the nature of the cause. When following continued irritation induced by dietetic errors, the mucous membrane is found to be opaque, swollen, and congested.

Treatment.—If acute gastritis be diagnosed, we should endeavour, in the first place, to ascertain the cause. If any poison has been administered, it will be necessary to treat the inflammation in accordance with the nature

of the toxic agent, and to administer the requisite antidote.*

Under any circumstances the food should be very restricted in amount, consisting of linseed gruel, or other non-irritating food, in small quantity. Opiates may also be given, especially if there be much pain.

2. CHRONIC GASTRITIS.—Etiology.—Chronic gastritis may be met with as the sequel of acute inflammation, but it usually follows dietetic errors.

In some instances it is traceable to irritation, caused by the presence of the larvæ of the œstrus equi, and may result from disease of the liver, or be dependent upon malignant disease of the walls of the stomach.

Crib-biting is also not unfrequently a cause of dyspepsia and chronic gastritis.

Symptoms.—The symptoms, for the most part, are those of chronic dyspepsia, which, though often spoken of as a functional disorder of the stomach, is frequently due to chronic inflammation of that viscus.

Some cases of frequently recurring colic and intestinal disorder are, no doubt, to be attributed to chronic gastritis.

Treatment.—Ascertain the cause, if possible, and restrict the diet.

The internal treatment will vary with the cause, but will in the main be similar to that recommended in chronic dyspepsia.

RUPTURE OF THE STOMACH.

Etiology.—Partial or complete rupture of the walls of the stomach is not uncommon among horses. It is mainly due to errors in dieting and work, and is more frequently met with among the heavier draught horses, which are

* *Vide* Chapter on Poisons.

especially subjected to irregularities of work and defective dietetic arrangements.

Rupture is especially likely to occur when a large amount of food is given after exhausting or prolonged work. Under these circumstances the food is especially liable to undergo fermentative changes from its longer retention in the stomach, owing to the slow and imperfect action of the gastric juice and the defective muscular movements of the walls. It is said to be more frequent in horses fed on bruised than on whole grains, especially when put to work after a full meal.

Rupture of the stomach is probably, in most instances, preceded by derangement and distension, or actual degeneration of the walls, consequent on chronic indigestion and other causes. It is rarely met with in young animals, but most commonly occurs in aged animals, especially when these have undergone severe exertion or have been overworked for a long period.

Symptoms.—Vomiting, or attempts at vomiting, generally occur in rupture of the stomach, but as it is not invariably present, and may proceed from other causes, as, for instance, from rupture of the colon, or other part of the intestine, and also from dilation of the cardiac orifice, it cannot be considered a diagnostic symptom. It is, however, more complete in rupture of the stomach, and in dilatation of the cardiac orifice, than in intestinal lesions. Rupture may be brought about by gradual distension of the walls, and without much pain, until the advent of peritonitis, which is set up by the escape of the contents of the stomach into the peritoneal cavity. Sometimes, however, rupture occurs suddenly, owing to the violent struggles of the animal in its paroxysms of pain, during the course of gastric disturbance, or in intestinal disorders.

The animal becomes uneasy, the countenance becomes

gloomy and dejected, and he looks anxiously round at his flanks. There is great weakness, and rapid prostration of strength. The pulse is feeble and fluttering, the respirations are short and quick, and there are frequent attempts at vomiting and regurgitation of the solid or fluid contents through the nostrils, and there may be profuse perspiration. In some cases the animal remains quiet for a time after the rupture, while in other instances the pain is intense, and the animal becomes delirious.

By some vomition is thought to be incompatible with rupture of the stomach, and is considered to be an antecedent symptom. It is, however, highly probable that vomition can and does occur after rupture of this organ, unless the rent be very extensive.

Prognosis.—Rupture of the stomach is fatal in the great majority of instances. In some, collapse and death follow in a few hours, while in others, where the rent is not so extensive, life may be prolonged for a couple of days or more.

Treatment.—Treatment is of no avail. If the diagnosis is clear, it is best to have the animal put out of his agony.

Section IV.

DISEASES OF THE INTESTINES.

ON SOME IMPORTANT SYMPTOMATIC CONDITIONS AND FUNCTIONAL DISORDERS OF THE INTESTINES.

CONSTIPATION.

Constipation, or torpid action of the bowels, may depend upon intestinal obstruction, which is treated of below, or upon diminished peristaltic action, or deficient intestinal secretions. The two latter are, in their turn, chiefly due to dietetic errors, though they may depend upon other

causes also. Retention of fæces, if not relieved, may lead to congestion or inflammation of the intestine.

Generally the abdomen is full and distended, but this is by no means a constant symptom. The motions are attended with difficulty, and straining or tenesmus is not unfrequent.

If the constipation continue unrelieved, the appetite becomes impaired, weakness follows, and the pulse becomes feeble and accelerated. In some instances a yellow gelatinous mucous secretion is discharged in cases of constipation depending upon retention and impaction of the fæces. This discharge is frequently mistaken for diarrhœa by the uninitiated, when in reality it is indicative of a costive condition of the bowels.

Treatment.—As long as the animal remains in good health, all that is necessary in constipation is a change of the diet for one of a more laxative nature. If the constipation is habitual, a moderate dose of aloes, followed by the administration of vegetable tonics, such as nux vomica, gentian, and several others, is efficacious in most instances.

When the irregularity of the bowels is dependent upon paralysis of part of the intestine, purgatives should not be given. In these cases, stimulants, with liquor strychnæ, will be found very beneficial. If the paralysis involve the large gut, the rectum is frequently found to be in a dry, dilated condition, and gives the impression, when the hand is introduced, of a large cavity with passive walls (Williams).

For the prevention of the recurrence of constipation, bran mashes and other laxative diet may be substituted occasionally for the more solid food, and nux vomica, gentian, and ginger may be given in the form of a ball three or four times weekly. If necessary, an occasional dose of aloes may be administered.

In cases where young foals are unable to void the meconium, enemas of oil will generally be found efficacious. If the bowels are not relieved by this means, a couple of ounces of castor oil may be given internally.

DIARRHŒA.

Etiology of Diarrhœa.—Diarrhœa is the general term applied to abnormal fluidity and increased amount of the alvine discharges. It is met with as a functional disturbance of various nature, or as a symptom in the course of general disease or extensive local changes in the intestinal tract.

The proximate causes of diarrhœa are excessive secretion from the intestinal walls, combined with increased peristaltic action. These conditions are, in their turn, either due to direct irritation of the mucous membrane from without, as, for instance, by food, foul water, parasites, or to indirect influences generated in the animal itself. As instances of the latter may be mentioned the diarrhœa which sometimes accompanies the specific fevers, and that which occurs in some structural changes in the liver, spleen and pancreas, and that resulting from disturbance of the nervous system, frequently reflex in nature.

Perhaps of all causes of diarrhœa, the most frequent in the adult animal is injurious and irregular dieting. Sudden changes in the diet, especially from a dry to a moist or laxative one, ingestion of medicinal substances, copious draughts of cold water, when heated after exposure to the sun's rays or exertion, and feeding immediately after severe work or exposure to cold and damp, may be mentioned as specially liable to induce diarrhœa.*

* Mr. Robertson mentions that diarrhœa is sometimes induced by the free use of potatoes ; but this practice is fortunately limited to certain parts of the country.

In plethoric horses with very little work, a small amount of exercise will often bring on an attack of diarrhœa.

Symptoms.—The fæces are semi-fluid, and either fœtid or without offensive odour. If the diarrhœa continues long unchecked, the animal loses flesh and the appetite fails.

In some instances, there is great prostration, abdominal pain is not uncommon, and the respirations become accelerated. Unless the nausea and pain be very marked, the pulse is not usually quickened. The special symptoms induced by the presence of parasites, among which the strongylus tetracanthus, a small nematode found chiefly in the cæcum and colon, is especially liable to cause diarrhœa, will be considered in the chapter on parasites.

Etiology, Symptoms, and Pathological Lesions of Diarrhœa in the Young.—In the young, diarrhœa in many instances differs from that of the adult, and has special characteristic features of its own.

The form of diarrhœa to which we refer is a specific intestinal catarrh, which though not contagious in foals, as it is probably in the bovine species, is nevertheless a far more serious affection than ordinary diarrhœa of the adult.

It owes its origin to defective sanitary arrangements, and also to changes in the quality of the milk. Such changes are traceable in some instances to the fact that the mare is worked hard during the day, and returns at night to her foal, which after its fast during the day is apt to take more milk than it can well digest.

The symptoms of this infantile diarrhœa usually appear during the first two or three weeks of life. The fæces at first are of a yellowish white colour, and there is little or no pain. In more advanced stages, or from the first in the

more serious forms, the fæces are acrid, and there is more or less abdominal pain, which may be very severe. If the disease continues, the foal ceases to suck, and loses flesh rapidly.

After death there may be no discoverable lesions, or there may be an accumulation of dark serous fluid in the abdomen, as well as several patches of ecchymosis on the peritoneal surface of the bowel. The mucous membrane of the bowel is infiltrated, and covered with a catarrhal discharge, while in some places superficial ulcerations, owing to the removal of the epithelium, may be found.

In some instances the liver is pale and bloodless.

Prognosis in Diarrhœa.—The prognosis is usually very favourable, but in infantile diarrhœa a fatal termination is not uncommon.

Treatment of Diarrhœa in Adults.—We should, in the first place, endeavour to ascertain the cause of the diarrhœa. If it proceed from irregularities in the feeding or in the work, these should be immediately rectified. In most instances medicine is not required unless the diarrhœa is excessive, or the pain and general disturbance very great. No cold water should be allowed; the animal should be kept quiet and warmly clad. The diet should be easily digestible, and linseed gruel or other demulcent drinks may be allowed. When the pain is very great, tincture of opium, spirits of chloroform in moderate doses, with a drachm of camphor, may be given three times daily in flour gruel. When prostration is very marked, and the pain severe, tincture of opium, sulphuric ether, and spirits of chloroform may be given three times daily, with a moderate amount of alcoholic stimulant, as port wine or brandy. Woollen cloths wrung out from warm water may be applied frequently to the abdomen, and stimulating liniments rubbed in in the intervals between the applications. When the

diarrhœa is due to hepatic engorgement or obstruction to the portal circulation, dilute nitric acid, with nux vomica and gentian, may be administered in the form of a draught twice daily.

Treatment of Diarrhœa in the Young.—It is well to commence the treatment of this form of diarrhœa with a laxative, such as castor oil, in order to expel irritant matter in the intestines.

A dose of tincture of opium may be given with the oil.

This treatment may be followed up by the administration of camphor and opium, with spirits of chloroform, three times a day, in water, or in strong decoction of tea.

When weakness is very marked, a little alcoholic stimulant may be added, and the hot cloths and stimulating liniments applied to the abdomen. Other remedies also found beneficial are carbonate of magnesia, catechu, Bael fruit, and prepared chalk.

COLIC.

Abdominal pain may arise from functional derangement of the intestinal canal, or it may be due to organic lesions of varying extent and nature. To the functional disturbance the term '*true colic*' is applied; while when depending on organic lesions this condition is sometimes spoken of as '*false colic.*'

True colic is of two varieties, which may be associated together. The one, termed 'spasmodic colic,' is due to spasmodic contraction of the muscular walls of the gut; the other, termed 'flatulent colic,' is owing to extensive gaseous accumulation in the intestine. It is said that the spasmodic form may terminate in intestinal inflammation. This is not at all improbable, but *nevertheless it is not easy to tell in such instances whether the colic may not be an early symptom of commencing inflammation.*

Etiology of Colic.—Colic may be due to dietetic errors, such as have been already mentioned as liable to induce dyspepsia, diarrhœa, constipation, and other disordered states of the system. Over-feeding, sudden changes in the diet, irregularities in the dieting, taking a large amount of food after a long fast, or after prolonged or severe exertion, food of inferior or unsuitable quality, are all potent agencies in the production of spasmodic intestinal contraction, as well as of flatulent distension, which may be associated with the spasm or occur independently.

Besides dietetic errors, there are many other causes of colic. This painful affection may be due to intestinal obstruction from mechanical displacement, and change of position of different parts of the intestine, or from impaction of calculi or other concretions of varying composition.

Not unfrequently colic depends upon the presence of animal parasites in the bowels, and sometimes also in neighbouring parts. Young animals especially, when badly fed and attended to, are more liable to colic from this source than older horses. Pain, when originating from the presence of worms, is usually of a recurrent type, and attended with progressing debility and loss of flesh. The presence of certain worms in the mesenteric arteries is, as we shall see in the chapter on parasites, also said to be a frequent cause of colic on the Continent, more especially in Germany. This parasitic affection, which is rarely met with in this country, sometimes even ends in death. Irritant poisons, when ingested, produce pain, sometimes very intense. Cold and damp also may cause intestinal disturbance, manifested by abdominal pain.

Lastly, as causes of colic are the group of organic diseases not only of the intestine itself, but also of the peritoneum, kidneys, liver, and pleura.

Among the organic diseases which may produce colic are:

disease, more especially abscess, of the mesenteric glands, which not unfrequently accompanies strangles and other suppurative diseases; chronic changes in the walls of the intestines, as, for example, thickening, degeneration or atrophy of the coats; malignant growths; intussusception and dysentery.

Symptoms of Spasmodic Colic.—The onset of spasmodic colic is generally more or less sudden. The horse shows signs of abdominal pain by looking round at his flanks, by restlessness, by striking at his belly with his hind feet, and in various other ways. He lies down, and rolls about from side to side. After a while he rises and eats a little, and soon perhaps a paroxysm of pain again attacks him.

In uncomplicated cases of colic the number of the pulse and respirations and the temperature are rarely elevated, except during the paroxysms of pain. The pulse is then much accelerated, and the respiration becomes hurried or sighing.

The attack may now subside, or gradually become more and more severe, the paroxysms becoming more continuous and the pain more intense.

The restlessness and excitability increase, or partial stupor supervenes, and the attack, if unrelieved, may end in death from continued pain or exhaustion, with varying complications.

In most cases of colic the bowels are constipated, and the fæces, if any are passed, are usually hard, and often coated with mucus. The urine is frequently retained, or passed in a jerky manner. Great restlessness, much pain, frequent pawing and looking to the sides, are said to be especially indicative of invasion of the small intestine, while stretching of the body, throwing of the head upwards, with curling of the upper lip and pressing of the posterior part of the body against the wall or stall-post,

more especially point to colic resulting from impaction of the large intestine.

Symptoms of Flatulent Colic.—Flatulent colic, due to distension of the intestine with gas, may be associated with spasm of the muscular coats, or it may occur independently of the spasmodic variety. The flatulent form is especially to be attributed to digestive disturbance depending on ingestion of food specially prone to undergo fermentation. This affection usually comes on suddenly. The horse is noticed to be very restless, and the abdomen distends and becomes tense, and gives a tympanitic note on percussion. The breathing is short, and chiefly thoracic, and the pulse is increased in frequency, and feeble. The extremities are cold, and there may be more or less delirium and vertigo. When the animal is inclined to lie down, he does not throw himself suddenly on the ground, as in spasmodic colic, but allows himself to fall more slowly and carefully.

If unrelieved, the continued distension and thoracic compression may lead to further circulatory and inspiratory disturbance, and death may result from asphyxia. Sometimes rupture of the colon or other part of the bowel, or of the diaphragm, is the cause of death.

Prognosis of Spasmodic and Flatulent Colic.—In severe cases of flatulent colic the prognosis is not so favourable as in the spasmodic form, but in ordinary cases the prognosis is very favourable in both varieties.

In all prolonged cases with great pain and restlessness there is danger of displacement or entanglement of the intestine; and when tympanitic distension is very great, or the struggles very violent, there is great risk of rupture of the large bowel or of the diaphragm.

Morbid Anatomy of Spasmodic and Flatulent Colic.—When colic is due to continued spasm, there may be found a peculiar degenerative change of both the mucous and sub-

mucous walls of the small and large gut. In other cases the change is of an atrophic nature.

In many instances, however, no change is to be found in the walls of the intestine, though these are not infrequently somewhat thickened.

In some cases parasites, or various kinds of concretions to which the pain was due, may be found after death.

When the colic depends on organic changes, corresponding lesions of varying extent are found.

Treatment of Spasmodic Colic.—In all cases of colic, except those in which diarrhœa is present, it is advisable to commence treatment by the administration of a cathartic. Barbadoes aloes is the best purgative in these cases, and is given preferably in the form of a ball, in doses of 5 to 7 or 8 drachms, depending upon the size and condition of the animal.*

In addition to the cathartic, enemas of water at about 100° F. should be given, and repeated at intervals of two or three hours if necessary. Some recommend the addition of aloes to the enema, or the substitution of oil for warm water in cases of impaction of the colon. We do not, however, believe that enemas of this kind are of greater efficacy than those of simple warm water. If the pain is severe, it is best combated by the administration of ʒi. ss. to ʒii. of sulphuric ether with ʒii. of nitric ether, which may be given in gruel or water, and may be repeated every two or three hours in the early stages so long as the pain continues unabated. Tincture of opium, or hypodermic injection of 40 minims of acetate of morphia (injectio morphiæ

* There is perhaps no drug in the pharmacopœia of which such varying qualities are found in the market as of Barbadoes aloes, and none in the buying of which greater care is required. Much of that sold is really unfit for use ; and it is well not to trust to balls of aloes sold ready made up by some. We believe that neglect of proper precautions in this particular is often answerable for very untoward results.

acetatis, B.P.), is recommended by some authorities. These opiates are also very efficacious in subduing the pain. The tinctura opii may be given in ʒij. to ʒiij. doses, and may be repeated if necessary after the lapse of two or three hours. It is not, however, generally necessary to repeat the hypodermic injection. A mixture composed of doses of tincture of opium and sulphuric ether, with a moderate dose of spirit of chloroform, is perhaps the most efficacious means of subduing the intense pain of colic. Some recommend Fleming's tincture of aconite; but we believe this remedy to be neither very efficacious nor necessary in the treatment of uncomplicated colic.

Hot fomentations and stimulating liniments may with advantage be applied to the abdomen.

Treatment of Flatulent Colic.—In these cases, in addition to the aloes, it is advisable to administer some remedy to dispel the accumulated gas. For this purpose, aromatic spirit of ammonia, turpentine, and assafœtida are perhaps the best. If there be much pain, an anodyne, as sulphuric ether, or tincture of opium, should be administered in addition.

French veterinarians largely practise puncturing the colon with a trochar, in order to liberate the accumulated gas. This measure is said by some to be attended with great benefit if performed early.

INTESTINAL OBSTRUCTION.

Etiology.—The causes of intestinal obstruction may be arranged under various headings:

1. *Causes acting within the gut itself*, such as impaction of fæces, or concretions of various kinds. Some of the latter are composed of vegetable matter, while others chiefly

consist of mineral substances, as phosphate of lime and magnesia.

The larger concretions are more frequently found in the pouches of the large intestine, from which they are liable to be displaced by the movements of the animal.

2. *Causes acting on the walls of the gut*, as inflammatory thickening, or stricture of any portion of the small or large intestine. These causes are rare in the horse.

3. *Causes acting on the gut from without*, resulting in altered position or displacement. As examples may be mentioned strangulation by bands of lymph, or, in rare instances, by mesenteric tumours, the neck of which wraps round the gut, and also twists and displacements of various kinds.

The colon may be found twisted round on itself, and sometimes the small intestine is entangled, owing to twisting or laceration of the mesentery. Not uncommonly a loop of the small, and sometimes of the large, intestine passes through an opening in the peritoneal covering.

Umbilical and inguinal herniæ may also cause obstruction, as also may *intussusception, or the passage of one part of the bowel into that immediately below it.* This condition is very rarely met with in the horse, and is of more frequent occurrence in the young than in adult animals.

General Symptoms of Obstruction.—The symptoms of intestinal obstruction, which may proceed from so many different causes, are very variable.

When the obstruction results from impaction of fæces, or from the presence of concretions, the symptoms are usually gradual and of an intermittent character.

The other forms are generally more sudden in their onset; but we know of no diagnostic symptoms peculiar to twists, intussusception, or strangulation of the intestines in the horse.

All of them may be associated with great enteric or abdominal pain, restlessness, sitting on the hind-quarters, small, frequent, thready pulse, accelerated respiration, cold extremities, distension of the abdomen, and collapse, ending in death from exhaustion.

In some cases it may be possible to detect an external hernia, when the diagnosis will be at once cleared up. The progress of the case will sometimes help us in forming a diagnosis; for example, if there be constipation, with frequent attacks of colic, the obstruction is probably due to impaction of fæces.

Treatment of Obstruction.—Anodynes, such as those recommended in colic, may be given, and repeated as often as necessary. Tincture of opium and sulphuric ether are perhaps the most efficacious. Enemata of warm water may be injected in full amount into the rectum, and hot fomentations, or woollen cloths wrung out from hot water, applied to the abdomen. The rectum may be explored also by the hand, with the object of removing obstructions when within reach.

In some instances tapping the distended bowel is recommended when the distension is very great.

The food should be of a laxative kind, and only allowed in moderation.

RUPTURE OF THE INTESTINE.

Rupture of the walls of the intestine is of more frequent occurrence in the colon than in the small intestine, and is due in most instances to impaction of fæces, or to excessive tympanitis, or to both these conditions associated together. It is readily intelligible that these disorders are especially liable to result where the walls of the gut are in an atonic or degenerated condition. Moreover, under these circumstances the muscular and other coats of the intestines are

least capable of resisting the effects of these disorders, and are thus especially prone to rupture.

Symptoms.—The symptoms of rupture are very variable, and not diagnostic. In most instances rapid exhaustion follows the intestinal lesion, wherever it may be situated.

In some cases collapse and death soon follow the occurrence, while in others life is not extinguished for several days. In many cases rupture is very difficult to diagnose from other severe affections of the bowels. When following impaction of fæces in the colon, or considerable dilatation of the walls of the gut, rupture is not unfrequently succeeded by relief. The restlessness and straining subside, and a period of calm follows until death. The countenance, however, is anxious, the pulse small, thready, and gradually becomes more and more imperceptible, the respiration is short and thoracic, and there is great disinclination to stir. In rupture of the colon the horse frequently sits on his haunches, and may attempt to vomit; but these symptoms cannot be regarded as diagnostic of rupture. Sitting on the haunches, indeed, is a very frequent symptom in twists and other forms of strangulation.

ENTERITIS — INFLAMMATION OF THE BOWELS.

Etiology and Varieties.—Inflammation of the bowels is in most cases limited to sections of the intestinal canal, though it may affect the tube throughout. It is more commonly met with in adults and in those in confinement than in the young and those running out at grass.

The late Mr. D. Gresswell used to recognise two distinct forms of enteritis in the horse, which, although presenting many symptoms in common, are in reality of a different nature.

The first variety may be termed *congestive*, or indeed *apoplectic*, from the rapidity with which the animal is struck down, as it were, and dies in a few hours. Of this variety the causes are not always apparent, but over-exertion, prolonged exposure to cold, drinking cold water when heated, and finally, washing with cold water while the animal is in a heated and perspiring condition, are among the chief which we are acquainted with.

The second variety, which may be termed *secondary enteritis*, is in most instances of not such a severe type, even though the extent of inflammation is in some cases very great. This form depends upon impaction of fæces, constipation, intussusception, the ingestion of irritative poisons, especially arsenic, various concretions, aggregations of parasites, and various other causes.

In addition to the above causes, enteritis may also supervene in some fevers and constitutional diseases. As an example may be mentioned the enteritis of pink eye, or pneumo-enteritis, as this form of influenza has been termed.

By some the cæcum and colon are said to be more frequently invaded than the small intestines, while other authorities affirm that enteritis has its seat more frequently in the ileum and jejunum.

Symptoms.—In some cases the symptoms of enteritis are gradual, while in others they are sudden in their onset. Not unfrequently the abdominal pain is preceded by general constitutional disturbance, shown by acceleration of the breathing, marked dulness, depression, and loss of appetite; while in other instances the inflammatory action is ushered in with marked shivering or rigors.

The belly is generally tender when pressure is applied.

The abdominal pain, unlike that of colic, is continuous, is more agonizing, and rarely has periods of intermission.

The pulse, at first quick, hard, and wiry, becomes in the

later stages still more accelerated, though of less volume, feebler, and gradually more irritable and imperceptible. In number the beats range from 70 or 80 to 120, or even higher.

In cases of simple colic the pulse is unaltered, except during the paroxysms of pain, whereas in enteritis it gradually becomes more and more disturbed.

The animal in his paroxysms of pain stamps and strikes at his belly, and when he lies down he may be observed to do so with greater care than in simple colic.

He often turns his eyes towards his flanks, and copious sweats bedew the body, and he groans in his agony. At other times he stands, so intense is the pain, almost motionless, with an expression indicative of acute suffering depicted on his countenance.

The surface of the body becomes cold and bedewed with moisture, the pupils dilate, and delirium and stupor may supervene.

The animal soon perhaps becomes more restless than ever, and wanders about the box, or casts himself down, and rolls about regardless of all obstacles.

Sometimes he will balance himself for a short time, with teeth clenched and limbs and ears icy cold, when he may suddenly fall, and die exhausted after severe struggles.

Sometimes before death an apparent improvement takes place; the horse stands quiet for a while, yet, though the breathing becomes quieter and the pain abates, and he takes a little food, the countenance maintains its haggard, dejected appearance, cold sweats bedew the body, and the pulse continues thready, and perhaps almost or quite imperceptible.

In still more advanced stages, if agony, pain, and intense inflammation have not already carried off our patient, he trembles continuously, the lips fall pendulously, the eyes

become duller and more amaurotic, the mouth becomes clammy, the breath perhaps fœtid, until at length he can hold out no longer, and death puts an end to his suffering.

Prognosis.—Enteritis is generally fatal. If, as happens in some rare instances, the acute symptoms abate after the lapse of a few hours, and the pulse regains in some degree its normal character, becoming fuller, softer, and slower, there is great hope of recovery.

Morbid Anatomy.—In the form of enteritis which we spoke of as congestive, the post-mortem appearances are very marked and characteristic.

The mucous membrane of the affected section of the gut is intensely congested, being of a deep purple or even black colour, and in many instances there is a copious effusion of blood in the intestinal canal. The mucous coat is much thickened, and can be easily separated from its connections with the underlying coats of gut. There is also considerable thickening of the submucous and subserous coats, which are intensely infiltrated with sero-hæmorrhagic effusion.

The colon is said to be more frequently the seat of this violent form of inflammation than any other part of the bowel.

In some cases, so extensive is the infiltration and thickening of the submucous tissue, and so intense is the inflammatory process, that this coat appears as a dark purple or black gelatinous mass of two inches or more in thickness, extending for varying lengths of the gut, and sometimes involving many feet of the intestinal tract.

Even though the amount of effusion into the gut be very great, and the contents themselves be fluid, the bowels usually remain inactive owing to paralysis of the muscular coats.

In other forms of enteritis the inflammatory process is

not of this marked character. The inflammation is usually more patchy in distribution. Coagulable lymph is effused on the surface, and afterwards may be voided with the fæces. The morbid appearances of enteritis caused by the ingestion of poisonous agents will be considered in a later chapter.

Inflammation of the intestines, which occurs in various fevers, such as influenza, is of a different character from the varieties already considered. In some instances the inflammation is attended in these cases with extensive serohæmorrhagic effusion into the submucous coat, while in others the exudation is a much less marked feature.

Sometimes ulcerated patches are observed in the mucous membrane, while in other cases the epithelial lining of the intestine is eroded in large tracts, leaving a rough granular surface. Not uncommonly dark gangrenous patches are encountered here and there on the mucous membrane.

Treatment.—We have said that in acute enteritis cessation of the peristaltic action results at the seat of the inflammation owing to paralysis of the muscular coats of the intestine. It is thus manifest that cathartic medicines, if administered, would cause increased peristaltic action and secretion above the seat of disease. They would thus only serve to further increase the irritation in the involved area by the passage of the fæces through it, while they could have no effect whatever on its functional activity, owing to the paralysis of the walls.

Furthermore, constipation, lasting a day or two, is not in itself a matter of very serious importance. Cathartics are therefore inadmissible in the treatment of this disease, being only calculated to promote the morbid processes in action. So far from our irritating the inflamed intestine for the relief of constipation, we should use all the means in our power to assuage pain and calm the movements of

the intestines. For this purpose no remedy is more efficacious than opium, or its alkaloid, morphia. Opium is best given in powder in the form of a ball, or in simple solution, and may be administered in doses of one, two, or three drachms in a suitable medium. Some authors recommend a draught of five minims of tincture of aconite, and half a drachm of opium. We prefer to give a larger dose of opium in the first instance, and have found that the addition to the mixture of two drachms of chloroform considerably enhances the beneficial result. Aconite is, however, also a valuable remedy in the treatment of enteritis, especially in those cases where bleeding, though admissible, is not practised.

After the administration of the opiate, if the pain still continue severe, 40 minims of the injectio morphiæ hypodermica (B.P.) may be injected under the skin every two or three hours if necessary.

Some veterinary surgeons prefer sulphuric ether for the relief of pain after the administration of the opiate, while others combine the two remedies. Sulphuric ether is without doubt a valuable remedy, especially in all cases where there is much flatulence, and we have found it of great value when given in addition to the hypodermic injection. Of the use of belladonna as a drug to be depended upon in enteritis, we cannot speak in high terms of praise.

Hot fomentations, by means of woollen rugs wrung out from very hot water, may be applied to the abdomen, and renewed every half-hour for three or four times, while the pain is very acute, and afterwards every hour or so. During the intervals some stimulating liniments may be well rubbed in, over the abdomen. Some practitioners recommend the application of a poultice of mustard, which is rubbed off in two or three hours, and followed up by the application of hot fomentations.

Enemas of tepid water should be given by means of the ordinary funnel apparatus, but it is not advisable to use any injecting syringe. If the enema cause much pain, it is well not to annoy the animal by continuing it. If the horse is inclined to drink, he may be allowed linseed gruel or tea, or oatmeal gruel.

After the abatement of the acute symptoms, no cathartics should be given, but we should endeavour to relieve the bowels by the administration of enemas and laxative diet, consisting of bran mashes, linseed, and oatmeal gruel. No hard food should be allowed on any account until all danger is over.

In highly plethoric animals, bleeding is indicated in the early stages, when the pulse is full, and is often very shortly followed by abatement of the acute symptoms. Blood should be abstracted in amount proportionate to the size and condition of the animal, and in moderation only.

It is our practice under any circumstances not to remove more than two or three quarts of blood.

The operation should not be repeated.

DYSENTERY.

Definition.—Dysentery (δυς, with difficulty; ἐντερον, the bowel) is an inflammatory disease of the intestine, chiefly the large one, accompanied by febrile manifestations and sometimes by abdominal pain, and is characterized by certain tissue changes which vary according to the intensity of the inflammatory process. The fæces are often mingled with blood, albuminous material, or shreds of disintegrated tissue.

Nature and Etiology.—Dysentery is of less frequent occurrence in the horse than in other animals, and may be

of an acute or chronic nature. It is not established as yet whether this affection can be communicated from one horse to another. In most cases dysentery occurs as an independent affection; sometimes, however, it supervenes on an attack of ordinary diarrhœa. Among the chief causes of this malady in the horse are:

1. *Malhygienic conditions*, such as overcrowding, vitiated air supply, and exposure to noxious emanations.

2. *Insufficient or bad food and foul water.*

3. *Exposure to cold and damp, overwork, and all other depressing agencies.*

4. *Malarial poison* arising from vegetable decay.

This is especially prevalent in low-lying marshy tracts and in shady places.

We have, as yet, no evidence that dysentery owes its origin to the entry of any specific germ into the system, already perhaps in an unhealthy depressed condition, but this seems not at all unlikely.

In the horse we have no proof that this disease ever arises from direct infection from another animal.

Symptoms.—The frequent passage of liquid fæces may first attract notice, or, in other instances, febrile manifestations, debility, and rapid prostration may precede the frequent alvine discharges.

Rigors are common throughout the disease in many cases. The pulse is accelerated, the internal temperature is elevated, thus distinguishing this affection from simple diarrhœa.

Sometimes the disease begins insidiously, and we may not suspect the true nature of the affection in the first instance. But as the disease advances the appetite becomes more impaired; there is great depression, thirst, tympanitis not unfrequently, general wasting, marasmus, and marked prostration.

The fæces are thin and watery and offensive, and are

sometimes voided with abdominal pain, and there is much straining and irritation about the rectum and anus.

If examined, the alvine discharges are observed to be made up of mucus, blood in varying degrees of coagulation and decomposition, small portions of indigestible matter, shreds of sloughing mucous membrane, and but very little true fæcal matter.

Prognosis.—In mild cases the prognosis is favourable, but in severe ones there is very little hope of recovery.

Morbid Anatomy.—Dysentery consists in inflammation of a diphtheritic character, being characterized by the formation of a greyish fibrinous material on the surface of the mucous membrane.

The large intestine is generally involved, the small being rarely affected.

The inflammation is at first of a patchy character, and may have a favourable termination, or ulceration follows, and dark-coloured irregular sloughs of variable extent are formed.

Treatment.—In the treatment of dysentery it is at first necessary to attend to the sanitary conditions.

The animal should be kept at rest, and the diet should be of an easily digestible, fairly nutritious, moist kind.

A small dose of oil may be given in the first instance, and this should be followed up by the administration of opium and antacids for a time. If the progress of the disease is not arrested, astringents, such as tannic acid, alum, perchloride or sulphate of iron, catechu, and bael fruit, may be tried. Nitrate of silver in solution, sulphate of copper, or acetate of lead with opium in the form of a ball, are recommended.

In order to act as a deodorizer and antiseptic, carbolic acid or hyposulphite of soda are indicated. Ipecacuanha* given

* Ipecacuanha is regarded as a specific in the treatment of dysentery in man.

in combination with opium is also said to be of great value in the treatment of this disease.

Section V.

DISEASES OF THE PERITONEUM.

PERITONITIS.

Etiology.—Peritonitis (περιτείνω, I stretch around), or inflammation of the serous covering of the abdominal viscera and walls, is not a common disease in the horse, though it may be set up by a variety of causes.

Idiopathic or primary peritonitis is met with as the result of exposure to cold and damp, and is of greater frequency as a sub-acute or chronic disease than as an acute affection. It is not uncommonly observed in this sub-acute or chronic form in young horses from exposure and insufficient diet.

*Traumatic peritonitis** is the form which follows the infliction of wounds of the abdomen, and various surgical proceedings, as castration and operations for hernia.

Sometimes inflammation of the peritoneum occurs as the result of injuries from within, as rupture or perforation of the organs in connection with it.

As instances may be given rupture of the walls of the stomach or intestine, or of the liver, spleen, or kidney, or of an abcess or hydatid in the substance, or in connection with the various abdominal organs.

Sometimes peritonitis follows bursting of the bladder from over-distension.

General or localized peritonitis may also be set up by propagation of inflammation from the bowels, liver, uterus, and mammary glands.

Lastly, *secondary or sympathetic peritonitis* occurs in some

* Traumatic peritonitis will be more fully considered in our Manual of the Theory and Practice of Surgery.

general diseases, for example, in certain specific fevers and constitutional diseases.

Symptoms.—The symptoms of peritonitis are very variable.

Acute idiopathic peritonitis is usually sudden in its onset, whereas the sub-acute or chronic form is slow and insidious in the development and manifestation of its symptoms.

The animal shows signs of abdominal pain, is restless, and paws the ground with its fore-feet. The pulse is small and wiry; the respirations hurried, shallow, and thoracic, and the temperature elevated.

The abdomen is tender, sometimes exceedingly so, when pressed upon, and is sometimes tympanitic.

The appetite is lost, and ascites may be developed.

In peritonitis following the infliction of injuries, the abdominal pain is generally more severe than in the idiopathic form.

Prognosis.—When general, peritonitis is fatal in most instances.

Morbid Anatomy.—As in the inflammation of other serous membranes, the stage of congestion is followed by the exudation of lymph, with or without the effusion of fluid, and by the proliferation, or 'germination,' of the endothelial cells which line the free surfaces of the peritoneal membrane.

The membrane becomes thickened, not, as a rule, uniformly, but especially in localized areas, in patches or streaks.

The lymph effused is yellow, forming a layer of variable thickness on the surface of the peritoneum, and leads to adhesions between the opposed surfaces. The adhesions become more coherent by age. The fluid effused may be small in amount, or abundant, especially in some forms, and

holds flakes of lymph in suspension. Sometimes it is purulent or contains blood.

Treatment.—In strong plethoric animals, blood may be abstracted in acute peritonitis, and internally Fleming's tincture of aconite, with full doses of opium, or the hypodermic injection of morphia is indicated in order to allay the pain, and to quiet the movements of the intestines.

Locally hot fomentations should be applied to the abdomen, and may be followed up by the use of stimulating liniments containing opium.

To the water or gruel which may be allowed freely to allay the thirst, solution of acetate of ammonium, or other saline medicine may be added. After abatement of the acute symptoms, half-drachm doses of digitalis with nitric ether, and solution of ammonium acetate may be given every three or four hours.

In chronic cases, good nutritious diet and careful attention to the sanitary condition are of first importance. Internally, vegetable and mineral tonics take the place of the bleeding and sedatives recommended in the acute form. If there be much abdominal pain, it may be combated by the administration of opium or morphia in small doses.

Stimulating liniments may be applied to the abdomen.

ASCITES.—Etiology.—The presence of serous fluid in the peritoneum is termed ascites (ἀσκος, a leather bag, or wine skin). This condition is often associated with anasarca, or general dropsy, and may be due to a variety of causes:

1. Chronic peritonitis.
2. Disease of the walls or valves of the heart.
3. Obstruction of the portal circulation, from disease of the liver.
4. Renal disease.
5. Enlargement, or other disease of the spleen.

6. Deposits and new formations on the omentum or mesentery.

Symptoms.—The symptoms are insidious when unassociated with some special disease.

The animal is dull, listless, loses flesh, and becomes weak and debilitated.

The abdomen becomes gradually and uniformly distended, and if there is much fluid present, dulness is manifested on percussion, and fluctuation may be felt.

The animal loses his appetite, and the pulse is weak and accelerated.

The symptoms, however, will vary much with the conditions on which the effusion depends, and in many instances œdema of the limbs and lower parts of the body follows.

The urine is scanty, and if the kidneys are diseased, contains albumen in greater or lesser amount. If the liver is at fault, the urinary secretion may contain bile-salts and bile-pigment.

Morbid Anatomy.—In many instances there is more or less thickening and structural change in the peritoneum. These changes are especially marked in ascites, resulting in chronic or sub-acute inflammation of the peritoneum.

In some cases there is no detectable change found in this membrane after death. In ascites depending on disease of the heart, liver, or spleen, or on morbid growths in connection with the peritoneum, lesions, varying with the nature of the affections, are found in the organ at fault.

The fluid varies much in amount. It is usually clear, slightly viscid, alkaline, of a yellow or green tinge, and contains a large quantity of albumen. When following inflammation, it is turbid and flaky.

Treatment.—The treatment of ascites chiefly depends upon the disease with which it is associated. We have

already spoken of the treatment of chronic peritonitis, and have only to add that if the fluid effused still remains unabsorbed, preparations of iron and vegetable tonics, with diuretics and the occasional administration of some purgative, are called for, in order to promote the general health and aid in the absorption of the fluid.

The iodide of iron, or the liquor ferri perchloridi, are more especially beneficial under these conditions.

When the ascites is due to cardiac mischief, in addition to the special treatment of the heart itself, diuretics and hydragogue purgatives are indicated.

If depending on renal disturbance, stimulating diuretics are inadmissible. Under these circumstances, perchloride or iodide of iron is very beneficial.

When the amount of the fluid effused is very great, and does not diminish after the employment of medicines, *paracentesis of the abdomen* may be performed, and repeated, if necessary. This operation, however, rarely affords permanent relief.

MORBID GROWTHS IN CONNECTION WITH THE PERITONEUM.—*Hydatid cysts** and *cancerous growths* are sometimes developed in connection with the peritoneum.

Cancerous growths of the peritoneum nearly always spread from some neighbouring organ, and very rarely appear primarily in this serous membrane.

Fatty growths and fibroid tumours are not uncommonly met with in connection with folds of the omentum and mesentery, and in other situations. Some of these tumours are pedunculated, and are thus liable to strangulate the intestine by entwining their necks around it.

These tumours cannot be diagnosed during life.

* *Vide* Chapter on Animal Parasites.

Section VI.

DISEASES OF THE LIVER.

GENERAL PHYSIOLOGICAL AND PATHOLOGICAL CONSIDERATIONS.

Diseases of the liver are of far less frequent occurrence in the horse than in man, but they are nevertheless of great importance, owing to the many and varied functions performed by this organ in health.

The liver has at least three important functions.

In the first place, it is concerned in the elaboration and secretion of bile. Secondly, amyloid and saccharine substances, brought to it by the portal vessels from the alimentary canal, are converted into glycogen.

Glycogen ($C_6H_{10}O_5$), or animal starch, is a substance resembling dextrin, and is similarly convertible into sugar by the action of albuminoid ferments. It is formed and stored up in the hepatic cells, whence it is removed as required by the hepatic veins, and is thus distributed throughout the system to be used as required.

It is used partly for the maintenance of the bodily heat, which is generated during its oxidization into carbonic acid and water, and partly to take an important share in the growth, development, and functional activity of cells, and also probably in the development of the blood corpuscles.

Glycogen is in all probability convertible into fat in the system.

Thirdly, the liver is concerned in the reduction of various albuminous matters brought to it into simpler compounds.

The latter include glycogen, leucin, tyrosin, uric acid, hippuric acid, and urea.

Of the clinical features of hepatic disease, jaundice, local pain in the region of the liver, manifestation of colicky pain,

persistent pain in the off-shoulder, ascites, congestion and enlargement of the spleen are among those most commonly met with.

From a *comparative pathological* point of view it is most interesting to observe how closely these manifestations of hepatic disorder correspond with those presented by man, in whom the lameness in the right fore-limb is represented by pain in the right shoulder.

Pain, indeed, may be the primary factor in the production of this kind of lameness in the horse.

CONGESTION OF THE LIVER—INTERSTITIAL HEPATITIS.

Nature and Varieties.—Congestion or engorgement of the liver with blood, or with biliary secretion, is probably the most common of the hepatic diseases of the horse, and is of three varieties, (1) active, (2) passive, and (3) biliary.

1. *Active Congestion.*—Active congestion of the liver always accompanies the process of digestion in health, but it is apt to exceed the normal limits if the food be supplied in excess, or if it be of a too highly stimulating nature.

2. *Passive Congestion.*—Passive congestion is the form most commonly met with, and is due to obstruction of the circulation through the hepatic and portal veins. This variety of congestion is more especially associated with obstruction of the blood-flow, depending on dilatation or valvular disease of the heart, or on disease of the pulmonary organs.

3. *Biliary Congestion.*—Biliary congestion, or over-filling of the small bile-ducts with secretion, may arise from active or passive hyperæmia, or from both. It is due to obstruction in the ducts from the presence of parasites or other foreign bodies, or to inflammation of the mucous membrane. It usually gives rise to pain, and is sometimes associated

with epizootic diseases. When influenza is accompanied by this form of congestion of the liver, it is termed *bilious* or *biliary influenza.*

Etiology.—The chief causes of the various forms of hepatic congestion are, previous pulmonary or cardiac diseases, specific fevers, dietetic errors, chills from exposure, and over-exertion of plethoric animals after a full meal.

Symptoms.—Slight jaundice, evidenced by the yellow tinge of the conjunctivæ, is usually a noticeable feature, especially in the variety described as biliary congestion.

Signs of digestive disturbance, such as loss of appetite and irregular action of the bowels, are usually manifested. Febrile symptoms are rarely present, unless the congestion be associated with some specific fever.

The extremities are cold, and pressure over the region of the liver causes pain.

The urine is generally scanty, and is more highly coloured than normally, and not unfrequently contains bile pigment.

Morbid Anatomy.—When congested, the liver is often considerably enlarged, from the great amount of blood it contains. This is especially the case in passive hyperæmia of this organ. The term nutmeg liver is applied to the peculiar variegated appearance of the surface on section which is found in cases of long-standing mechanical congestion.

This condition is characterized by a large accumulation of blood in the hepatic veins, which become dilated and thickened; by atrophy of the hepatic cells in the central portions of the lobules, and by increase of the inter-lobular connective-tissue.

The impediment to the return of blood causes the atrophy of the hepatic cells, and the formation of granules of pigment. When examined microscopically, the lobules are

seen to be made up of broken-down cells and pigment granules.

On section, the liver presents a mottled appearance, the centre of the lobules being a dark red colour, whilst the peripheral parts are of a yellowish white.

The veins are found to be much dilated and filled with red cells.

Not infrequently, blood extravasations and ruptures, owing to the engorgement, are met with as the result of repeated attacks of congestion and inflammation of the liver.

When these ruptures or extravasations are very extensive and serious, they are manifested during life by collapse or sudden pain. When less extensive there may be no diagnostic symptoms, beyond perhaps a little abdominal pain, which may not attract our attention.

Treatment.—In cases where the congestion is not due to cardiac or pulmonary disease, and where the onset is sudden, bleeding the animal in proportion to his size and condition is recommended.

In congestion of the liver associated with the specific fevers, however, it is not advisable to abstract blood.

The blood-letting should be followed up by the use of moderate doses of aloes, and the subsequent administration of salines, such as sulphate of soda, or sulphate of magnesia.

When the congestion is gradually developed and is dependent on dietetic errors, or want of proper exercise, it is of first importance to restrict the amount of food, and attend to the sanitary conditions. In addition, salines, such as those above recommended, may be given in the drinking-water.

When associated with some specific fever, the treatment need not materially differ from that of the primary malady.

External applications, such as stimulating liniments or warm-water fomentations, are recommended by some authorities in the treatment of cases of congestion.

In order to prevent the recurrence of these affections of the liver, the diet should be limited and regular exercise should be enjoined.

HEPATITIS—INFLAMMATION OF THE LIVER.

Hepatitis (ἡπαρ, the liver; itis), or inflammation of the liver, may involve the capsule (Glisson's capsule), and its prolongations into the tissue of the gland, or it may affect the secreting structures of the organ.

1. Inflammation of the Capsule of the Liver.—Peri-hepatitis.—This disease is occasionally met with as a sequel or accompaniment of pleurisy, and sometimes it may follow inflammation of the peritoneum.

This disease cannot be diagnosed during life.

2. Inflammation of the Gland Tissue—Hepatitis.—Inflammation of the glandular structure is rarely met with in the horse. It may occur in a localized or in a diffused form, and its causes are in many cases similar to those of congestion.

In hot countries it assumes an epizootic form, especially about the end of summer, and it is almost always connected with disease of the other abdominal organs (Gamgee). Hepatitis is very difficult to diagnose with certainty from acute congestion.

Localized hepatic inflammation, terminating in the formation of abscess, is rare in the horse, but secondary abscesses are frequently formed in the liver in pyæmia, and may be very numerous.

It seems probable that some cases described as hepatitis are in reality instances of active congestion of the liver.

CIRRHOSIS OF THE LIVER—CHRONIC INTERSTITIAL HEPATITIS.

Etiology.—Cirrhosis (χίρρος, yellow), though of more frequent occurrence in the horse than acute inflammations of the liver, is far more rarely met with than in man.

Very little, indeed, is actually known of its causes in animals. In man, as is well known, the chief agent in its production is abuse of alcoholic liquors, and, with the exception of syphilis, no other cause of this condition has been *with any certainty* ascertained.

Defective and insufficient food-supply are said by some to be the causes of cirrhosis in the horse; but, though this may be so, nevertheless in some undoubted cases good food has been supplied in abundance.

By others, cirrhosis is said to follow repeated attacks of inflammation and congestion of the liver.

Symptoms.—The symptoms of cirrhosis are not as yet clearly known. Among the most important are ascites, congestion and irritability of the intestinal mucous membrane, dyspepsia, impaired appetite, and loss of flesh. Sometimes indications of jaundice may be manifested, and the liver may be tender on pressure. There are rarely any febrile symptoms.

As a result of the congestion of the intestinal canal, gastric and intestinal hæmorrhage may follow, though this is a very rare occurrence.

Morbid Anatomy.—In the early stage of cirrhosis the liver is enlarged, from the gradual increase in the connective-tissue in the organ.

Cirrhosis consists in a cellular infiltration of the interlobular tissue, and in the development of a more or less highly organized fibroid structure, which is supplied with new blood-vessels from branches of the hepatic artery.

Gradually the fibroid structure contracts, and thus the liver becomes smaller, and altered in form. The hepatic cells undergo fatty degeneration, and the lobular vessels and ducts are compressed and obliterated. In consequence of this, the portal circulation is obstructed, and ascites and congestion of the abdominal viscera result.

Treatment.—In the first place, the diet should be carefully attended to. Internally, sulphate of sodium or of magnesium may be given twice daily, with vegetable tonics.

If there be ascites, diuretics may be administered in addition to salines and tonics.

In some cases it may be necessary to perform paracentesis abdominis.

FATTY LIVER.—Fatty changes in the organs of the body are due to infiltration of the tissue with fat, or to degeneration or conversion of the albuminous substance into fat.

Fatty liver is strictly an infiltration, but the term is used to include both these forms of change.

In the horse, fatty changes in the liver are traceable to overfeeding and want of sufficient exercise.

The blood becomes loaded with fatty materials, which are first deposited in the hepatic cells of the circumference of the lobules, and then gradually extend inwards.

The liver thus usually becomes enlarged, and increased in weight, and its margin becomes rounder and its surface smoother. It is of a soft or doughy consistence, and shows a pale, greasy surface on section.

In this affection there is no ascites or jaundice, but the functions of the liver are impaired if the fatty change is considerable.

The only treatment consists in regulation of the amount of exercise and limitation of the diet, and in the occasional administration of a purgative.

LARDACEOUS LIVER.—Lardaceous degeneration of the liver is sometimes met with after death, but there are no clinical symptoms by which it may be positively diagnosed during life.

The characters and appearances presented by lardaceous degeneration of the various organs of the body has already been described (page 31). When involving the liver, the conversion of the albuminous substances into lardacein usually commences in the *middle zone of the lobules*, corresponding with the distribution of the hepatic artery.

Lardaceous degeneration of the liver is often accompanied by similar changes in other abdominal organs, and is attended by general impairment of health and progressive loss of flesh.

HYDATID TUMOURS AND OTHER MORBID GROWTHS OF THE LIVER.

Hydatid Tumours.—Hydatid tumours (ὑδατις, a vesicle) are usually due to the development of the cysticercal embryo of the Tænia Echinococcus of dogs.

They are not of common occurrence in the horse, though the liver is perhaps more frequently their seat than any other organ.

The relationship between a cysticercus and its fully developed mature tapeworm is described in the Chapter on Animal Parasites, to which the reader is referred for the full history.

A hydatid tumour consists of a primary sac or bladder, with thick, soft, transparent, and laminated walls, the innermost layer of which is formed by a delicate cellular membrane. The sac (mother-cyst) is filled with clear, limpid fluid, in which are numerous daughter-cysts. On the inner surface of these secondary cysts there are situated many little prominences, which consist of small vesicular

bodies, each surmounted by a head. These are the immature forms of the Tænia Echinococcus.

The tumours do not present any symptoms by which their presence can be diagnosed.

Cancerous Tumours.—When infiltrated with nodular or diffuse so-called cancerous tumours, the liver is enlarged, and may be altered in shape.

Some of these morbid growths spread rapidly, while others extend much more slowly.

The growths are of rare occurrence in the horse.

Melanotic Deposits.—Melanotic growths are occasionally found in the liver in circumscribed patches, or in diffused masses.

Lymphoid Tumours.—Lymphoid growths, when occurring in the liver, are often numerous. They occur as smooth nodules of varying size.

JAUNDICE—ICTERUS.

Jaundice is, strictly speaking, a symptom of many affections in which the tissues of the body are dyed yellow; but it is of such importance as to merit a separate description. Jaundice (*jaunir*, to become yellow), or icterus (ἰχτερος, a yellow bird), is so called from the yellow discolouration of the conjunctivæ, skin, and visible mucous membranes, due to impregnation with the biliary pigment.

Etiology.—Jaundice may be due to—

(*a*) *Excess of bile in the blood.*—This may be caused by obstruction to the flow of bile into the duodenum, leading to absorption of the secretion by the blood, which thus becomes contaminated.

Obstruction may depend on catarrhal inflammation of the mucous membrane of the ducts, or upon the presence of foreign bodies in the bile-duct.

Excess of bile in the blood may also depend upon excessive secretion, which also leads to absorption.

This occurs in hepatic congestion.

(*b*) *Suppression of the secretion of bile, or interference with its metamorphosis.*—This may occur in certain specific fevers and general disorders; for example, influenza and pyæmia.

Symptoms.—The symptoms of jaundice are rarely well marked in the horse. As a result of impregnation with bile pigment, the tissues soon become stained yellowish; the conjunctivæ, skin, and the visible mucous membranes more especially show the yellow staining.

The urine becomes more deeply coloured from the presence of bile pigment, and the motions become drier and of lighter colour, from the deficiency of bile flowing into the duodenum.

Unless jaundice follows a general or specific disease, there are usually no febrile manifestations, and the pulse is in most instances not accelerated, and the temperature similarly remains normal.

In some cases loss of appetite and digestive disturbance are marked features, and depression, languor, and incapacity for exertion follow.

Itching and cutaneous eruptions on the skin are occasionally met with in severe cases.

Lameness in the off-shoulder is in some instances an accompaniment of jaundice.

Tests for Bile Pigment and Bile Acids :—

Gmelin's test for bile pigment in the urine.—Add a drop or two of strong nitric acid to a small quantity of urine on a piece of white porcelain, when, if there is bile pigment present, there will be a play of colours, passing through green, blue, violet, and red, into a dirty yellow.

Pettenkofer's test for bile acids in the urine.—Add to a small quantity of the urine, in a test tube, a very small quantity

of cane sugar, and afterwards pour in some strong sulphuric acid very slowly. If bile acids be present, there will be first thrown down a white precipitate of cholalic acid, and afterwards the whole of the liquid will become of a deep, beautiful purple colour. This reaction is facilitated by gently warming the side of the test tube.

Treatment.—When jaundice is developed in connection with a specific fever, no special treatment is required, unless the symptoms be of a very pronounced character.

In other cases the treatment depends upon the cause of the affection.

In the first place it is necessary to increase the amount of exercise, if this be insufficient, and to restrict the amount of food allowed.

Mild cases may require no further treatment beyond the occasional administration of saline purgatives, or moderate doses of aloes.

When depending on hepatic congestion, the treatment is similar to that already described under this affection.

If jaundice be attended with much debility and languor, vegetable tonics, with doses of nitro-hydrochloric acid, may be given three times daily.

Besides the remedies already mentioned, chloride of ammonium and calomel, in moderate doses, are also recommended in some forms of jaundice.

Section VII.

DISEASES OF THE SPLEEN.

General Physiological Considerations.—Very little is known of the functions of the spleen. In this organ white blood corpuscles appear to be formed, and red corpuscles to be broken down. The spleen receives an abundant supply of blood, and undergoes considerable changes in

volume. It has movements of contraction and expansion, which are slow and rhythmical. The spleen may be exterminated in animals and man without any serious ill effect. Hypertrophy of some of the lymphatic glands, and red marrow of bones follows, and these tissues probably take on a vicarious function. The blood coming from the spleen is said to contain a larger number of white cells than that entering it.

In diseases in which the spleen is enlarged, the blood contains a large excess of white cells.

Diseases of the spleen are very rarely, if ever, diagnosed in the horse during life, though various pathological changes are not uncommonly met with in making post-mortem examinations.

Enlargement of the Spleen.—Enlargement of the spleen is of rare occurrence, except when depending on abnormal growths, as lymphadenoma and melanosis.

Lymphadenoma.—Lymphadenoma of the spleen is sometimes found as greyish masses of about the size of a cherry, and sometimes still larger.

These formations in the spleen are said by some to be accompanied by leucocythæmia (λευκος, white; κυτος, a cell; and ἁιμα, blood) in many cases.

There are no diagnostic symptoms by which these tumours can be recognised during life.

They are, however, not unfrequently accompanied by anæmia, anorexia, and loss of flesh.

When examined microscopically, we have found these tumours of the spleen to be composed of an *adenoid reticulum*, or network of fine homogeneous fibrils, the meshes of which are completely occupied by lymph corpuscles.

Other Morbid Growths of the Spleen.—Cancer, melanotic growths, and lardaceous degeneration are sometimes met with after death, but cannot be diagnosed during life.

CHAPTER VIII.

DISEASES OF THE URINARY SYSTEM.

THE URINE IN HEALTH AND IN DISEASE.

As renal diseases are intimately connected with alterations in the character of the urinary secretion, a knowledge of the chemical composition of the urine, and of the alterations it may undergo in disease, is of great importance.

THE URINE IN HEALTH.—Healthy urine is an amber-coloured fluid, varying in composition in different animals, but containing in all analogous products of nitrogenous waste and various salts.

In the carnivorous animal it is a clear watery fluid of acid reaction. In herbivorous animals it is either alkaline when secreted, or very soon becomes so; and is often cloudy, depositing a sediment on standing.

These differences in the character of the urine depend upon the nature of the food of the animal.

If a herbivore be fed solely upon a nitrogenous diet, the urine becomes acid, as also it does in certain diseases. In inanition, also, where the body may be said to feed upon its own tissues, the urine of herbivora likewise becomes acid. Similarly, if a carnivorous animal be fed upon vegetables, the urine becomes alkaline and cloudy.

The urine of the mammalia contains a large amount of water, and in the horse has a specific gravity of 1030—1050.

According to our own investigations the average of a large number of estimates of the specific gravity of the urine is 1041·2.

The actual amount of urine passed per day is estimated by Colin at 15—25 litres (44·033—61·646 pints); this estimate gives an average 20 litres a day. This amount is somewhat in excess of the average—18·91 litres—found by us in a number of cases.

The quantity of water in 1,000 parts of urine has been estimated at 880—930 (Von Bibra and others).

The chief constituents of the urine are water, products of nitrogenous decomposition, of which urea is the most important, hippuric and uric acids, inorganic salts, extractives and colouring matters.

The actual amount of solids in 100 parts of urine of the horse we have estimated to average 8·955.

Urea.—Urea is by far the most important of the solid constituents of the urine. It is derived from the disintegration or metamorphosis of albuminous and albuminoid substances of the food and of the tissues, and contains most of the nitrogen which was originally incorporated with the substances from which it is derived.

Urea, or carbamide ($CO(NH_2)_2$) is isomeric with ammonium cyanate (NH_4CNO, or CH_4N_2O). It is excessively soluble in water, from which it crystallizes in white silky needles or transparent four-sided prisms, the ends of which are often formed by one or two inclined planes.

It has a bitter saline taste, is colourless, soluble in alcohol, but insoluble in ether.

Nitrate of urea may be obtained in considerable quantities by the addition of excess of nitric acid to concentrated urine.

This salt crystallizes in rhombic prisms or plates, having a characteristic imbricated arrangement and mother-of-pearl lustre.

Oxalic acid similarly throws down oxalate of urea, which crystallizes in rhombic plates or prisms.

In the urine of the horse the urea bears a proportion of about 1 to 8, or 1 to 9, of all the solids (Gamgee).

The amount of urea secreted bears a definite relation to the amount of food taken into the system. It is largest when the food is albuminous and abundant; it is rapidly diminished by starvation, and gradually by a diet containing a large proportion of amylaceous or starchy matter.

It is augmented by increased amount of water drunk, and by the ingestion of sodium chloride and ammonium salts.

Urea is probably to a great extent formed in the liver, and its amount is most likely largely influenced by the condition of the hepatic cells and by the activity of the hepatic circulation.

Urea is also increased in many febrile disorders. In acute pneumonia and in laminitis we have especially noted a very large increase in the amount passed, and have already spoken of the amount in azoturia. (*Vide* 'Azoturia.')

Retention of urea in the blood in interference with the secretion of urine is associated for the most part with organic renal disease, though it may also be due to obstruction in the ureters. Urea, when thus retained in the blood, gives rise to an aggregate of symptoms termed **Uræmia.**

Uric Acid.—The urinary secretion of herbivorous animals contains little or no uric acid ($C_5H_4N_4O_3$), as this substance is represented in them by hippuric acid. It occurs, however, in the urine of man, and in that of almost all carnivorous animals. Uric acid can be crystallized from urine containing it by adding hydrochloric acid, which by decomposing the urates sets the uric acid free.

The most common forms of uric acid crystals are the whetstone-shaped, and the sheaf-like bundles of flattened needles.

Uric acid is insoluble in alcohol and ether, and very feebly soluble in water.

Hippuric Acid.—Hippuric acid ($C_9H_9NO_3$) occurs in very small proportion in the urine of man (less than ·1 per cent.) and carnivora, but is present in abundance as alkaline hippurates in that of herbivorous animals. Hippuric acid is a beautiful white crystalline substance, soluble in 400 parts of cold water. It is also soluble in ether and alcohol.

When the quantity of urea in the urine is small, that of hippuric acid is large, and vice versâ. It varies from five to fifteen parts in 1,000 of the urine of the horse (Bibra).

Hippuric acid crystallizes in long, transparent, four-sided prisms, and may be readily obtained by boiling the urine of an herbivore and adding hydrochloric acid.

It is noteworthy that in all animals in which the urine contains much hippuric acid, as in the horse, indigo is also present in relatively large quantities (Sanderson).

Hippuric acid is said to be formed in the system from the benzoic acid, or bodies which yield benzoic acid, which is ingested.

This acid is also said to appear in the urine of man and herbivora whenever benzoic acid ($C_7H_6O_2$) enters the system, glycin (glycocoll) being taken up and water given off.

Extractives.—The extractives found in the urine of the horse are creatin, creatinin, lactic acid, benzoic acid, and several others.

Pigmentary Matter. — The pigmentary or colouring matters of the urine of the horse are not of much physiological importance.

Inorganic Salts. — The urine of carnivora contains sulphates, chlorides, and phosphates; that of herbivora contains a large percentage of carbonates, with some

sulphates and chlorides. The urine of the latter, therefore, is found to effervesce on the addition of an acid. The chief metallic elements found in the urine are sodium, magnesium, calcium, and potassium.

Von Bibra found in 1,000 parts of the urine of the horse 42·20 parts of salts, which were composed of 78·37 per cent. of carbonates of lime, magnesium, potassium, and sodium; 13·04 per cent. of sulphate of potassium; 6·94 of chloride of sodium, and ·055 of silica.

The deposit which settled on allowing the urine to rest, being analyzed three times by Von Bibra, was found by him to have the following composition:

	I.	II.	III.
Carbonate of Calcium	80·9	87·2	87·5
Carbonate of Magnesium ...	12·1	7·5	8·5
Organic Material	7	5·3	4·3

THE URINE IN DISEASE.—In some diseases one or more of the normal constituents of the urine are increased, while in other affections they may be diminished in amount. Moreover, various abnormal constituents may be found in the urine in disease which are not present in health.

INCREASE OR DIMINUTION OF THE NORMAL CONSTITUENTS.—The amount of urine passed varies considerably, even in health. Some diseases, however, are especially characterized by an increase, while in others the secretion is much diminished. The urine is always scanty in febrile conditions and in acute inflammation of the kidneys, and may be almost or quite suppressed, as in collapse.

In diabetes insipidus, or hydruria, and in certain other conditions, as when diuretics are taken, the urine is in-

creased, owing to the excess of water eliminated through the kidneys.

The amount of urine may be much increased without any augmentation in the amount of solid constituents.

The specific gravity of the urine, however, is, as a rule, in inverse proportion to the amount passed; but under some conditions, as in diabetes mellitus, the density is high, although the urine is abundant. Again, we cannot conclude that there is an excess of solids present when the specific gravity is high, for this may be due to deficiency of water.

The re-action of the urine is normally alkaline, though in certain diseases it sometimes becomes acid. The reaction may be tested by litmus paper. If alkaline, urine turns red litmus blue; if acid, it turns blue litmus red.

We have already said that the amount of urea excreted may be increased or diminished under various abnormal conditions.

ABNORMAL CONSTITUENTS PRESENT IN THE URINE IN DISEASE.—Blood.—Blood is sometimes present in the urinary secretion, and may be derived from the kidneys, ureter, bladder, or from the urethra.

The passing of blood in the urine is termed **hæmaturia.**

When the blood has its source in the kidneys, it gives a general red or brownish smoky hue to the urine; which, on standing, deposits a brownish red sediment, in which blood-casts are often present.

In hæmorrhage from the bladder, the blood is far less intimately mixed with the urine, and often appears only toward the end of micturition, while the urine is of a pinkish or blood-red colour, and is associated with the symptoms of vesical disease.

Where blood is derived from the urethra, it is voided independently of micturition.

The causes of renal hæmorrhage may be arranged in two classes :

1. Local pathological lesions, viz.,

(*a*) Those due to congestion or inflammation of the kidneys.

(*b*) Cancerous or melanotic growths, or the presence of calculi in the kidney.

2. Symptomatic causes, or those depending upon some general or constitutional disease.

Blood-casts, or moulds of the uriniferous tubes formed by the coagulation of the blood in their lumina, are not uncommonly found in the urine in cases of renal hæmorrhage.

Blood in the urine may be detected by the presence of the red corpuscles seen in it when examined microscopically.

The appearance of the cells varies somewhat according to the specific gravity of the urine ; if it be high, the cells are small and shrivelled ; if it be low, they are large, transparent, and circular.

The urine may also be tested by the addition of a drop or two of tincture of guaiacum and a few drops of ozonized ether to a small portion in a test-tube, when, if blood-pigment be present, the ether which collects at the top will be coloured blue.

Urine containing blood is of necessity albuminous.

Albumen.—The presence of albumen in the urine in any quantity is of pathological significance. It may be found in the urine from structural changes in the kidney in renal congestion, or may be due to the presence of blood or pus.

Albumen, when present, may readily be detected.

If the urine be alkaline, as it frequently is, it is first necessary in testing it to add a few drops of acetic acid to a small portion in a test-tube, in order to slightly acidify it. After this the urine should be boiled, when, if albumen be present, it will be coagulated, forming opaque flakes, which gradually subside to the bottom of the tube.

Albumen may also be tested for by nitric acid. This test consists in pouring into a test-tube a small quantity of strong nitric acid, and then pouring very slowly down the side of the tube a little of the urine to be examined. If albumen be present an opaque white cloud forms immediately where the two fluids come into contact.

A saturated solution of picric acid also precipitates albumen, and picric acid tablets are now sold by chemists, and afford a ready method by which the presence of albumen may be detected.

Bile Acids and Bile Pigment.—Under the diseases of the liver, we have already spoken of the occasional presence of bile acid and pigment in the urine, and have given tests by which their presence may be recognised.

Grape Sugar.—Grape sugar is found in the urine in diabetes mellitus, under which disease tests by which this substance may be detected have been mentioned.

The fermentation test may also be applied. If a little yeast be added to urine containing sugar, after standing for some time in a warm place, it will ferment, with the disengagement of carbonic acid.

Pus and Mucus.—Pus is sometimes found in the urine, which is albuminous in proportion to the amount of pus present. If examined microscopically, the sediment is seen to contain pus-cells which are very similar to white blood-cells. They are, however, lifeless, and have no amœboid movements, and are characterized by the presence of several nuclei.

SECTION I.

DISEASES OF THE KIDNEYS.

RENAL CONGESTION. — Etiology and Symptoms.— Hyperæmia, or congestion of the kidneys, may be active or mechanical. It is not often met with in the horse.

Active congestion constitutes the first stage of inflammation of the kidneys, or nephritis. It sometimes occurs as the result of acute fevers, and may be induced by the ingestion of certain irritants, as turpentine and cantharides, or by the action of cold and damp.

Passive or mechanical congestion depends upon venous obstruction from pulmonary or cardiac disease.

When examined, the kidneys are seen to be enlarged and engorged with blood.

If the congestion, especially when mechanical, be of long duration, important structural changes of a chronic inflammatory nature may follow.

Renal congestion is usually attended with a scanty secretion, and if very severe, there may be a quantity of albumen, blood, and a few renal casts, but otherwise there are few or no general symptoms by which this affection may be diagnosed.

Treatment.—When dependent upon other affections, renal congestion must not be treated apart from the general disease.

In other cases, rest, laxatives, and the application of warm fomentations to the loins are all that is required.

INFLAMMATION OF THE KIDNEYS.—NEPHRITIS.—Inflammation of the kidneys, or nephritis (*νέφρον*, the kidney), is of rare occurrence in the horse.

In *acute* nephritis, as in all acute inflammations, the *cellular* structure of the organ is chiefly involved, whereas when this passes on to the *chronic* form the changes are more especially marked in the *interstitial* tissues.

Inflammation of the pelvis of the kidney, or pyelitis (*πυελον*, pelvis), except when resulting from the irritation of a calculus, is of rare occurrence in the horse. Professor Williams mentions a case of pyelitis in a foal two days old, which died of acute suppurative arthritis.

Symptoms of Nephritis.—When nephritis supervenes on some acutefever, the urine may be noticed to become more scanty and highly coloured, and may contain albumen, and renal epithelial casts. Blood, and fibrinous or sanguineous casts may also be present.

The internal temperature is raised still higher, and the skin becomes harsh and dry.

There is usually little or no pain on pressure of the loins in this form.

In nephritis due to other causes, the symptoms are somewhat different. There is considerable fever with colicky pains, the attack resembling colic, attended by fever. The pulse is quickened, and the artery is full, hard, and firm. The respirations are short and accelerated, the bowels are constipated, and thirst is increased. In some instances there is stiffness, tenderness in the loins, and arching of the back, but these symptoms are not invariably present.

The most characteristic features of nephritis are the scanty elimination or total suppression of urine and the desire to micturate frequently. The animal strains violently, but may be unable to pass more than a few drops of urine. This is highly-coloured, and if examined, albumen, blood, and renal casts may generally be detected in it.

Where, however, no urine is passed, the continued fever, abdominal pain, and frequent, though ineffectual, straining to micturate, are indicative of inflammation of the kidneys. But cases of nephritis are met with sometimes, in which the only symptoms are suppression of the urine (ischuria), with febrile manifestations, and of this several instances have come under our notice.

In other cases there are signs of pain manifested by lying down and rolling about, while at other times the seat of pain is pointed at by the animal turning round and endeavouring to bite at or scratch the loins.

If suppression of the urine be prolonged, other symptoms supervene, owing to retention of urea in the blood. The accumulation of urea, as we have already indicated, gives rise to symptoms of uræmic poisoning. The animal becomes partially unconscious, but there is no loss of motor power.

Retention of the urine may, however, continue for several days without the manifestation of coma.

As the urea gradually accumulates in the blood, there is a peculiar ammoniacal odour of the breath, which is also emitted from the skin and fæces.

The bowels are usually constipated, and muscular twitchings are not uncommon.

In the stallion, there may be retraction of the testicle on the affected side.

Sequelæ.—Chronic nephritis may be left as a sequel after the subsidence of an attack of acute inflammation of the kidneys, or it may manifest itself as an independent affection.

Atrophy and degeneration of the kidneys may follow acute nephritis.

Morbid Anatomy.—In acute nephritis, one or both kidneys are involved. They are much enlarged and engorged with blood. The surface is smooth, and the capsule is thin and non-adherent.

On section the colour is of a dark dusky red, and the malpighian bodies are of a still darker hue. At a later stage the cortical part of the kidney becomes paler or mottled, and contrasts strongly with the dark-red bases of the pyramids.

If a section be examined microscopically, the uriniferous tubules are found to be filled with epithelial cells in various degrees of disintegration, fibrinous material, and blood. These changes are more especially marked in the convoluted tubes.

The blood-vessels are distended with blood. In chronic nephritis supervening on the acute variety, the appearances may differ but little from the above, except in the great paleness of the cortex. In all long-standing cases the interstitial-tissue becomes more involved in the pathological change. It first becomes increased in amount, and afterwards contracting, leads to contraction and atrophy of the kidney.

In acute pyelitis there is redness, swelling, and ecchymosis of the lining mucous membrane, and a discharge of mucus and pus.

In chronic pyelitis the mucous lining is thickened, and the pelvis is dilated and may be filled with pus, which is due to the irritation caused by one or more calculi. Abscesses are sometimes found in the cortex of the kidney as the result of pyelitis. They may also be formed as the result of chronic inflammation of the kidney itself, or from an injury.

Treatment.—In acute nephritis caused by cold, fatigue, or exhaustion, if the pulse be strong, bleeding is very beneficial. Those cases, however, supervening various fevers, or due to injury, do not bear depletion so well.

The bowels should be freely acted upon by the administration of some purgative. Aloes is perhaps the best cathartic in the horse. Oil is not so reliable. The looseness of the bowels should be kept up by the administration of saline purgatives, such as sulphate of sodium, or sulphate of magnesium, which may be given in the water. Some authors recommend the administration of opium, if the pain be great, but this remedy is best withheld in the treatment of nephritis.

The pain may be relieved by the application of woollen cloths wrung from hot water, or of linseed-meal poultices over the loins.

The poultices and fomentations may be medicated by the addition of belladonna or digitalis, with great advantage.

The diet should be laxative, consisting of linseed and oatmeal gruel, and bran mashes.

When the acute symptoms have abated, salts of iron and vegetable tonics are indicated.

LARDACEOUS DEGENERATION OF THE KIDNEY.—Lardaceous degeneration is very rare in the kidneys of the horse.

On pathological grounds it should be distinguished from chronic nephritis. The appearance of the kidney resembles that of other organs similarly affected.

MELANOTIC TUMOURS AND HYDATIDS.—Melanotic tumours are sometimes, though rarely, found in the kidney of the horse.

Hydatids of the echinococcus have been met with in the kidney, though they are of far commoner occurrence in the liver.*

RENAL AND URETERAL CALCULI.—Renal calculi are rather rare in the horse, though they are met with in this animal more frequently than in any other. Ureteral calculi are of such rare occurrence as to require no special description here.

Renal calculi are of a brownish-white, sometimes bluish colour, of irregular ovoid or nearly spherical shape, often

* About a month or two ago we had under treatment an aged cart-horse suffering from chronic renal disease. The horse had great difficulty in passing water, and this contained abundance of pus and mucus. After death the post-mortem examination revealed about thirty hydatid cysts in the right kidney. They varied considerably in size, one being as large as a cocoanut; the others varied from the size of a walnut to that of a pea. In the left kidney there were also more than a dozen of these cysts, which were for the most part of less size. There were none present in the liver, though a great number were found in the substance of the psoas muscle. The right kidney was considerably atrophied.

nodulated, and always more or less rough, owing to the irregular deposition of carbonate of lime. They are placed in cysts with firm parietes, which contain a quantity of thick urine. They are distinctly laminated, and often numerous (Gamgee).

The deposition of calculi in the kidneys does not often cause symptoms by which their presence can be diagnosed. The urine, however, is often charged with earthy matter, and there may be occasional internal hæmorrhage and colicky pains, especially after the animal has been severely worked or exhausted.

Analysis of Renal Calculi of the Horse (Furstenberg).

	1.	2.	3.	4. *Corral Form.*	5. *Round Form.*		6. *Round Form.*	7. *Laminated Form.*	8. *Sedimentary Form.*
Specific gravity ..	2·263	2·241	2·121	2.283	2·194		2·193	1·918	1·6
					Centre of Stone.	*Surface.*			
Carbonate of lime	84·20	69·4	85·90	41·30	78·46	73·24	38·45	40·57	89·40
Carbonate of magnesia	1·56	9·1	5·20	11·04	4·66	5·37	38·29	16·28	3·35
Oxalate of lime ..	5·31	6·5	Trace	28·46	15·11	17·22	14·49	..	..
Phosphate of lime	..	..	..	7·03	..	..	..	..	..
Chloride of calcium	..	Trace	..	..	..	..	..	..	..
Sulphate of lime ..	..	Trace	..	..	..	..	..	..	..
Organic material ..	6·05	5·5	7·70	10·37	0·56	2·92	7·65	40·72	4·35
Water, Iron and loss	2·88	9·5	1·20	1·80	1·21	1·25	1·12	2·43	2·90
Total	100·00	100·0	100·00	100·00	100·00	100 00	100·00	100·00	100·00

Treatment of Renal Calculi.—Renal calculi are best treated by the administration of hydrochloric acid.

The diet should be well regulated.

In some instances calculi become impacted in the ureter, inducing more or less rapid degeneration of the kidney and

suppuration in its substance. Their impaction and passage along the ureters cause relapsing, excruciating pain.

Section II.

DISEASES OF THE BLADDER.

CYSTITIS, OR INFLAMMATION OF THE BLADDER.

Etiology.—Cystitis is rarely met with in the horse.

It may be caused by the administration of certain irritants, as cantharides or turpentine, or by the absorption of cantharidine from a blister. It may also be set up by the presence of calculi or morbid growths.

Symptoms.—The animal becomes restless, and shows signs of pain; he makes frequent attempts to pass water, but micturition is attended with difficulty and pain.

There are also signs of constitutional fever. If the cystitis be due to cantharides, or croton oil administered by the mouth, the alimentary canal throughout is more or less inflamed. If cantharides be absorbed from a blistered surface, the effect is chiefly upon the urinary organs, causing hæmaturia and sexual excitement.

Morbid Anatomy.—The mucous membrane of the bladder is variously altered in proportion to the intensity of the inflammation.

The membrane-lining usually presents an appearance of ordinary acute or chronic catarrhal inflammation, attended with more or less secretion of mucus or muco-pus. The urine often becomes rapidly ammoniacal. In very severe cases the mucous membrane may become ulcerated.

Treatment.—In cystitis arising from absorption of irritant poison from a blister, the latter should be at once washed off.

Internally, opium and hyoscyamus, or belladonna, are

indicated. The bowels should be regulated by laxative diet, and demulcent drinks should be allowed.

If there be much pain, and marked constitutional disturbance, moderate blood-letting, followed by mild purgatives, is recommended by some. Hot fomentations applied to the abdomen are very beneficial also in alleviating the pain when severe.

In chronic cases, hyoscyamus, buchu, and copaiba, with saline purgatives, are recommended by some. By others, mineral acids, with iron, are said to be very beneficial in the treatment.

RETENTION OF URINE.

Etiology.—Retention of urine is caused by spasm of the neck of the bladder in colic. It is a common complication in paraplegia, in enlargement of the prostate in aged animals, in urinary or urethral calculi, and in cancer of the penis. It may also be caused by obstruction at the opening of the urethra, prolapse of the uterus or vagina, or by accumulation of fæces in the rectum, or of dirt in the sheath (Williams).

Inability to urinate may be due to other causes, as, for example, when an animal cannot rise in azoturia, laminitis, or paralysis.

Paralysis of the bladder, due to over-distension of the walls by urine, may be the cause of retention. This sometimes occurs when an animal is not able to micturate for a lengthened period during a long journey.

Symptoms.—The animal, although making frequent attempts to micturate, is unable to pass water.

Sometimes a few drops may be passed; at other times, none at all.

In some cases there is a continual dribbling of the urine. In retention, if the bladder be examined by the hand passed through the rectum, it will be found distended.

Treatment.—The treatment of retention is essentially surgical. Careful catheterization is necessary in most cases, though in some instances pressure on the distended bladder by the hand passed through the rectum will suffice.

INCONTINENCE OF URINE.

By incontinence we mean an inability to retain the urine.

In some instances retention is accompanied by incontinence of urine, which continually dribbles, owing to the great distension of the bladder. In other cases incontinence may be due to the presence of calculi, or to paralysis of the sphincter vesicæ.

When due to over-distension, the incontinence must be treated by catheterization.

When due to paralysis, nux vomica and vegetable tonics with injection of cold water into the rectum are indicated.

CHAPTER IX.

DISEASES OF THE REPRODUCTORY SYSTEM.

HYSTERIA.

During the period of œstrum in the mare, symptoms resembling those of hysteria have been observed.

In two cases described by Professor Williams, the symptoms manifested were—clenching the jaws, grinding the teeth, difficulty in deglutition, and squinting, together with some degree of trismus, and tonic alternating with clonic spasms of the voluntary muscles.

Kicking with the hind-legs in a most persistent and uncontrollable manner, stamping with the hind-legs, and other indications of great restlessness and excitement, are very marked in some cases. The vulva, when examined, is found to be in a hyperæsthetic condition, being opened and closed in rapid succession. The urine, which at first is excessive, becomes more scanty and high-coloured.

In one of the cases recorded, the animal was prostrate and rigid, and appeared as though in a state of trance.

In the case of a mare recently under our notice, the animal kicked violently and almost continuously, and there was great sexual excitement. The urine was pale, watery, and abundant. The mucous membrane of the vulva and vagina was highly congested.

Treatment.—Depletion was practised in the former two cases, one of which recovered. The bleeding was followed by administration of cathartics. In the case before our notice, a moderate dose of aloes was first given, and was followed up by the administration of full doses of potassium and sodium bromide, with tincture of assafœtida three times daily. A laxative diet of bran-mashes and linseed-gruel was ordered. The mare recovered completely in a few days.

PARTURIENT FEVER.

Definition.—Parturient fever is a slight febrile condition occurring usually in from twenty-four hours to three days after parturition, and usually terminating in recovery.

Etiology.—The fever affects animals of all ages, but is of more frequent occurrence after the first parturition.

It is caused by cold, or by malhygienic conditions; and it may also be induced by dietetic errors.

Symptoms.—The pulse is quick and full, and the respirations are accelerated. The bowels are constipated, and the temperature is raised somewhat.

The visible mucous membranes are injected; the secretion of milk is arrested, and the lacteal gland is harder and hotter than normally.

Treatment.—A mild oleaginous purgative should be given, and should be followed up by the administration of liquor ammoniæ acetatis and nitric ether.

The diet should be laxative, and restricted in amount.

ACUTE METRITIS.

Acute metritis, or acute inflammation of the uterus, occurs within a few hours to three or four days after parturition, and is an extremely fatal disease.

Etiology and Nature.—The causes of acute metritis are fatigue previous to parturition, injuries to the uterus and

vagina during delivery, and retained placental membranes. This disease may also be induced by exposure to cold or other debilitating influences.

Acute metritis may be regarded as due to pyæmia, or septicæmic poisoning, depending generally on absorption of septic matter from the parturient canal. Secondary foci of inflammation appear in the brain, lungs, and other organs.

When due to the retention of the placental membranes, the disease depends upon the decomposition of coagula and pieces of the placenta within the uterus, and the absorption of the products of their disintegration into the blood.

Symptoms.—The animal is uneasy, and loses its appetite. The milk becomes very nearly, or quite suppressed.

If any can be squeezed out, it is a bluish tint, and thin and watery, or it may be a reddish-brown colour.

The vulva is small and contracted, and the vaginal mucous membrane sometimes of a deep red or purple hue, with petechial spots and purple streaks.

There is violent straining, and the discharge of a dark-coloured, fœtid, decomposing fluid.

The pulse is feeble, thready, and accelerated; the heart's action disturbed; the surface of the body cold.

The animal shows signs of abdominal pain, and sometimes manifests symptoms of inflammation of the feet.

As the disease progresses the animal gradually falls, the pulse becomes feebler and feebler, and the breathing hurried and thoracic, until at length complete unconsciousness sets in, the animal dying in from twelve hours to two days or more after the onset.

Morbid Anatomy.—There is diffuse inflammation of the mucous membrane of the uterus, which soon extends to the uterine veins.

The inflammation extends to the peritoneum and intestines, and tends to spread over a large surface.

The inflammatory exudation is dark coloured and abundant.

That which is effused into the uterine cavity causes great irritation and straining, and is discharged through the vagina.

Examined microscopically, the discharge is found to consist of blood globules, pus, and much granular matter intermixed with shreds of lymph.

Treatment.—In the first place it is essential that the uterus and vagina be washed out by injections of Condy's fluid, carbolic acid, or hypo-sulphite of sodium, very largely diluted with warm water.

This is necessary in order to prevent the further absorption of septic matter.

The strength of the animal should be supported by the internal administration of alcoholic and other stimulants.

If the temperature be very high, salicylate of sodium or quinine should be given in addition.

The abdominal pain may be relieved by opium, and hot fomentations to the loins and abdominal walls.

INFLAMMATION OF THE OVARIES, OVARITIS, OOPHORITIS.

Inflammation of the ovaries is very rarely met with in the equine species, and consequently very little is known of this disease.

Acute ovaritis, according to Percivall, appears to give pretty much rise to the same symptoms as denote peritonitis and metritis.

Chronic ovaritis is not marked by any appreciable symptoms, but lays the foundation for tumours of con-

siderable size, which may exist without disturbance of function.

Acute ovaritis may terminate in suppuration, or pass into the chronic stage, but generally ends in resolution.

Sometimes the ovarian tumours may burst into the abdomen, and thus cause death. The late Mr. D. Gresswell noted three cases of ovaritis in the mare, and of these one was acute and the other two of the chronic form.

CHAPTER X.

DISEASES OF THE NERVOUS SYSTEM.

GENERAL REMARKS ON THE STRUCTURE AND FUNCTIONS OF THE NERVOUS SYSTEM, AND ON THE LOCALIZATION OF NERVOUS DISEASES.

We do not purpose here to enter into the anatomy and histology of the nervous system, but to cursorily review some general points which are of especial importance in the study and in the localization of the diseases of the nervous system.

The nervous system is made up of two ultimate factors, nerve-fibres and nerve-cells, and these are intimately associated together.

The white matter of the brain and of the spinal cord is composed of nerve-fibres imbedded in a ground substance, or neuroglia. The grey matter of the brain and spinal cord is made up of nerve-fibres and of nerve-cells, or 'ganglion cells,' as they are termed, similarly imbedded in the neuroglia.

The neuroglia itself consists of three different kinds of elements—(1) a homogeneous, transparent, semi-fluid *matrix*, which in hardened sections appears more or less granular; (2) a network of very delicate fibrils, '*neuroglia fibrils*,' which resemble elastic fibres; (3) small branched nucleated cells

interwoven into the network of the neuroglia fibrils. These are termed the *neuroglia cells* (Klein).

Now, the nerve-cells alone are capable of exhibiting independent nervous activity.

They are the receivers of impressions and the generators of nerve-force, and therefore the true physiological nerve-centres are exclusively confined to the grey matter. The nerve-fibres only serve to conduct impulses to and from the nerve-centres with which they are in connection.

Cerebro-Spinal System.—On a vertical section of the hemispheres of the cerebrum, each convolution of the brain shows a white centre composed of medullated nerve-fibres and a grey cortex.

The white matter of the convolution is arranged (1) as a central mass—the centrum ovale, from which the lamina of white matter for each convolution branches off, and (2) the commissure of white matter between the two hemispheres, *i.e.*, the corpus callosum, and the anterior commissure (Klein).

The centrum ovale consists of (*a*) tracts of medullated nerve-fibres which connect the convolutions of the same hemisphere with one another, and (*b*) those passing between the convolutions on the one hand, and the thalamus opticus, the pons varolii, and the medulla on the other.

From the whole of the cerebral surface, therefore, white fibres radiate to the large ganglia at the base.

A special part of the corpora striata, termed the internal capsule, chiefly receives filaments from the anterior half of the brain, and these are concerned in the transmission of motor impulses. They pass through the 'crusta' of the crura cerebri, and thence along the motor tracts of the cord. The other ganglia at the base mainly receive fibres from the posterior part of the brain, and are chiefly concerned

with sensory functions. These fibres pass through the tegmentum of the crus, and thence along the sensory tracts of the medulla and cord.

The motor fibres, on reaching the medulla oblongata, decussate or cross over to the opposite side, pass down the anterior and lateral columns of the cord, and emerge by the anterior roots of the spinal nerves. The sensory fibres enter the cord by the posterior roots of the spinal nerves, cross immediately to the opposite side, thence pass upwards to the medulla, and onwards in the way above indicated.

If, therefore, one half of the cord below the medulla be destroyed, loss of motion on the same side as the lesion and loss of sensation on the opposite side, would follow. Destruction of one half of the brain would cause loss of motion and sensation on the opposite side to that of the lesion. Pathological observation and recent experimental researches have shown that combined movements uniformly follow excitation of certain areas of the cortical surface, and that destructive lesion of the same parts is followed by paralysis of special groups of muscles. But for further particulars of these special centres we must refer the reader to Dr. Ferrier 'On the Functions of the Brain,' or to Dr. Foster's 'Physiology,' in which a very clear account of the localization of cerebral functions is fully given.

Seeing that the structure of the brain is far less complex in the horse than in man, and that it forms, as the following table of Colin will show, a much lower proportion to the rest of the body, diseases of the nervous apparatus are of far less frequent occurrence in the equine species than in ourselves.

Table indicating the weight of the encephalon and spinal cord as compared with that of the body (Colin).

Animal.	Weight of Body.	Weight of Brain.	Weight of Medulla and Pons.	Total Weight of Encephalon.	Weight of the Spinal Cord.	Weight of Cerebellum.	Weight of Encephalon and Spinal Cord.	Relation between the Weights of the Encephalon and Body.	Relation between the Weights of the Cerebro-spinal Axis and Body.
Stallion ..	382·0	494	46	616	304	76	920	:: 1 : 620	:: 1 : 415
Gelding ..	380·0	559	39	675	300	77	975	:: 1 : 563	:: 1 : 389
Mare ..	408·0	510	34	615	269	71	684	:: 1 : 663	:: 1 : 461
Ass.. ..	175·0	316	24	385	159	45	544	:: 1 : 454	:: 1 : 321
Hinny ..	186·0	466	31	564	198	67	762	:: 1 : 329	:: 1 : 244

The anterior portion of the surface of the brain appears to be the supreme organ of the cerebro-motor processes or impulses, whereas the posterior portion of the cerebrum is similarly related to sensation. The part of the cerebrum anterior to the motor tract is chiefly concerned with the manifestation of intelligence.

Besides the ganglia already mentioned at the base of the brain, which include the corpora striata, the optic thalami, the corpora geniculata, and the corpora quadrigemina, there are collections of grey matter constituting a series of subordinate centres, closely succeeding each other, from the corpora quadrigemina above to the termination of the cord below, each either giving origin to motor or receiving sensory fibres. Each centre is also capable of independent action in connection with reflex or automatic movements. The most important function of the cerebellum is the co-ordination of voluntary movements. It has been held that it presides over the reproductory functions, but several facts oppose this view. M. Leuret showed, by taking the averages in ten stallions, twelve

mares, and twenty-one geldings, that the weight of the cerebellum is greatest in the latter.

	Average.	Highest.	Lowest.
Stallions..	61	65	56
Mares.....	61	66	58
Geldings..	70	76	64

Experimental lesions of the cerebellum always induce disorders of equilibrium, but never of impairment of sensation or actual loss of voluntary muscular power, and Flourens inferred, therefore, that it controlled and harmonized the various muscular movements, while Foville attributed the manifestations to loss of muscular sense.

Sympathetic System.—The chief function of the sympathetic system is the control of the involuntary muscle-fibre of the body.

If the branches of the sympathetic supplying the muscle be stimulated, contraction is caused, while section of the nerve is followed by relaxation of the fibres.

In this manner the calibre of the bloodvessels and the amount of the blood supplied to various organs and tissues is regulated.

In addition to this indirect influence of the sympathetic upon the nutrition of the tissues, it is very probable that it also exercises a direct influence on the functional activity of the cells in secreting glands and elsewhere.

The sympathetic system appears to have its supreme centre in the medulla, or rather in its floor; but it is intimately interwoven with the spinal system, for each spinal nerve receives branches from and transmits branches to the neighbouring sympathetic ganglion.

CONVULSIONS AND SPASMS.

The term convulsion, in its most extended sense, indicates any spasmodic contraction beyond the control of the animal.

Convulsions may affect a single muscle, or a group of muscles, a limb, or the whole or one half of the body.

They vary in intensity, from a mere 'tremor' or 'trembling' of a single muscle to the most violent contortions of the whole body.

Convulsions may be considered under three groups :

1. **Tremors.**—Tremors may accompany voluntary movements, or may occur independently. The tremors from cold or mercurial poisons are good examples of the latter.

2. **Choreic Movements.** — The disease termed chorea furnishes the most typical example of this kind of convulsion. Choreic movements are characterized mainly by their abruptness and irregularity, and by the fact that when engrafted on any voluntary movement, they interrupt its progress by a series of contractions which are not then necessarily limited to the limb or organ which is making the effort (Bristow).

3. **True Convulsions.**—They may arise from direct irritation of the nerve-centres, and also from irritation in remote parts.

Convulsions are clonic or tonic.

Clonic convulsions are intermittent and irregular, and those occurring in epilepsy will serve as an example. Tonic spasm consists in more or less sudden muscular contraction, and is exemplified in tetanus and strychnia poisoning.

MOTOR PARALYSIS—PARESIS.

By paralysis is meant the impairment of that power which the various motor centres normally exercise over the movements of the muscles.

The term 'paresis' is often used of the slighter forms of this condition.

Paralysis of the voluntary movements, which we now propose to consider, may be complete or incomplete.

Motor paralysis has special designations, in accordance with the parts invaded:

1. **General Paralysis.**—Is very rare in the horse, occurring only as a temporary condition in certain diseases of the cerebral structure, in cerebral congestion, in effusion into the basal ganglia, and in injuries and diseases of the anterior part of the cord and medulla oblongata.

2. **Hemiplegia.**—Hemiplegia strictly implies a paralysis of all the muscles of one half of the body; but in this perfect form it is never met with in man or animals.

Hemiplegia is very rarely observed in horses. It is usually the result of hæmorrhage or tumours affecting one half of the brain.

As a result of such changes, the angle of the mouth may become relaxed and the facial expression on the same side as the pathological lesion may be changed. The tongue may be protruded, and there is difficulty in drinking and in mastication. Deglutition is not impaired.

The animal may not lose the power of standing, but the power of progression is much affected.

The paralysis of the body is on the opposite side to that on which the cerebral lesion occurs. This is owing to the decussation of the motor fibres in the medulla oblongata.

The horse may regain power in a few days, but recovery is rare.

Those cases which end in recovery probably depend upon temporary inflammatory action, or other causes.

3. **Paraplegia.**—Paraplegia commonly depends on disease of the spinal cord, and denotes a defect or loss of power in all parts below the level of the lesion.

This affection is the commonest form of paralysis in the horse.

Causes.—The causes of paraplegia are:

1. *Central Nervous Lesions.* — These are due to injury, as fracture, or to diseases of the vertebræ, involving the spinal cord, or to disease of the cord itself.

2. *Reflex, or Functional Causes.* — Functional paraplegia depends upon alterations in the quality of the blood, as in lead poisoning, azoturia, and after feeding upon rye-grass; or it may depend upon intestinal or other disturbances of the system.

Paraplegia resulting from the ingestion of rye-grass (enzootic paraplegia) will be considered fully below.

4. Local Paralysis.—Local paralysis is very rare in the horse, and is usually due to injury of a nerve from pressure by badly fitting harness or by some abnormal growth. It may also be due to disease of the nerve at any part of its course, from its connection with nerve-centre to the organ supplied; but paralysis resulting from this cause is very uncommonly met with in the horse. The most common form of local paralysis in the horse is that of *the branches of the facial nerve, by which the muscles of the face are chiefly supplied with motor fibres.*

In paralysis of this nerve the lips are pendulous, and the animal is unable to grasp his food and has difficulty in mastication.

HYPERÆSTHESIA, DYSÆSTHESIA, ANÆSTHESIA.—By **hyperæsthesia** is meant exalted sensibility; by **dysæsthesia**, persistent or painful sensibility.

Practically, exalted sensibility is scarcely, if ever, distinguishable from painful sensibility. The hyperæsthetic eye cannot bear light, and the hyperæsthetic skin is sensitive to the slightest pressure.

The term 'dysæsthesia' includes the sensations of itching,

numbness, burning, cutting, constriction, and various other kinds of pain. The agony of enteritis, and several other diseases of the bowel and other organs, are especially acute in the horse.

By **anæsthesia** is meant impairment or loss of sensation. Hyperæsthesia and anæsthesia are usually met with together, associated with motor paralysis.

COMA AND INSENSIBILITY.

Under this heading we do not purpose to discuss the affection spoken of as 'sleepy staggers,' sometimes termed 'coma,' but to consider coma in its more general and extended sense.

Coma is a progressive loss of function, which begins at the cerebrum, and is characterized at the outset by loss of consciousness, perception, and voluntary movement.

Automatic and reflex movements continue at first; but when the large ganglia at the base of the brain become involved, the capacity for automatic movement becomes more and more limited.

In fatal cases, the loss of function progresses, and gradually extends to the medulla, which is the seat of the respiratory, circulatory, and other centres.

Death then soon ensues.

The causes of coma are intense congestion or anæmia of the brain, altered conditions of the blood, and injury or compression of the brain.

STAGGERS.

To several affections of the horse the term 'staggers' has been applied at various times by different authors.

Indeed, there are at least as many as five distinct affections which have received this name, and there are many more of which 'staggers' is a feature.

Staggers may be due to *primary disease of the nerve-centres, or to influences affecting them from disease of other organs.*

Varieties:—

1. Mad Staggers.—The condition termed 'mad staggers' may depend upon various causes. In the first place frenzy, or violent uncontrollable fury, may be caused by inflammation of the membranes of the brain, or the brain itself, or of both together. The frenzy-stage, or stage of irritation, occurring in meningeal inflammation, though undoubtedly also present in some cases of pure cerebritis (*vide* next article), is not such a constant feature as in the former disease.

After the frenzy-stage, as the disease progresses, stupor, coma, and paralysis may supervene.

Mad staggers, or frenzy, may also be due to uræmic poisoning, to rabies, or to melanotic deposits in the brain.

Again, staggers resulting from engorgement of the stomach may be so severe as to admit of being termed 'mad staggers.' Staggers dependent upon functional disturbance is not followed by coma and paralysis, as it is when dependent upon encephalitis, nor is it accompanied by febrile manifestations, as in the latter case.

2. Stomach Staggers.—This affection is otherwise known as acute indigestion or impaction of the stomach, and has already been described. Stomach staggers is sometimes termed 'sleepy staggers,' or 'coma.' Sleepy staggers, or coma, manifested by staggering gait; slow pulse and deep respirations may be, however, and frequently is, dependent on actual cerebral mischief.

3. Grass Staggers.—This affection is due to eating rye-grass, and will be described under 'Reflex or Enzootic Paraplegia.'

4. Epilepsy.—Epileptic attacks have been confounded with staggers depending upon secondary cerebral disturbance due to reflex irritation.

5. Megrims and Vertigo.—This affection, which is usually

due to irregularity or disturbance of the cerebral circulation, has been described as staggers.

Section I.

DISEASES OF THE BRAIN.

ACUTE INFLAMMATION OF THE BRAIN AND ITS MEMBRANES.

Synonyms.—Encephalitis, cerebritis, phrenitis, meningitis.

Acute inflammation of the cerebrum, or hemispheres of the brain, termed cerebritis, encephalitis, or phrenitis, is usually associated in the horse with meningitis, or inflammation of the meninges or coverings of the brain; and we therefore describe these two affections together.

Etiology.—Inflammation of the brain and of its coverings is usually due to direct injury, as blows on the head, or to disease of the bones of the cranium.

This inflammation sometimes supervenes in some of the specific fevers, and may also be due to exhaustion or exposure to the heat of the sun.

Of epizootic cerebro-meningitis, or cerebro-spinal fever, we have already treated.

Symptoms.—Practically, it is almost impossible to distinguish between inflammation of the brain itself and inflammation of its membranous coverings, and, indeed, as we have already intimated, these two affections are usually associated together.

When the membranes of the brain are primarily affected, the symptoms are characterized by the suddenness of their onset. They consist at first in great excitement, with convulsions or spasms, and are followed by a stage of depression.

Encephalitis is not characterized by such a sudden onset,

and is said to be marked by no frenzy or excitement, but from the first exhibits lowered or depressed functional activity.

But in some instances of encephalitis, at any rate, the stage of depression is preceded by one of irritation, as in meningitis. We ourselves have observed two instances, corroborated by the post-mortem examination, in which localized encephalitis was ushered in by the manifestation of violent and uncontrollable frenzy.

Nevertheless, great excitement and convulsions are especially characteristic of meningeal inflammation as distinguished from pure encephalitis.

The symptoms of meningo-encephalitis vary much in accordance with the extent of the tissue inflamed, and its seat.

The temperature in this affection is high, and the febrile manifestations are strongly marked. The appetite is capricious, and the bowels are confined.

The animal is very intolerant of its head being handled or pressed upon, and the skin and mouth are hotter than natural.

The eyes are staring and the pupils are contracted, though in the later stages of the disease they become widely dilated. The pulse is accelerated; the artery is full, firm, and hard, and the respirations are quickened and irregular.

The urine is diminished in amount, and is of darker colour than in health.

The horse moves to and fro restlessly, and his body is sometimes bedewed with perspiration.

Occasionally muscular twitchings and general or local hyperæsthesia are manifested.

The stage of excitement is of variable duration, and the symptoms manifested in it differ widely in intensity.

It is followed by the stage of depression, which is characterized by stupor and coma, caused by more serious involvement of the true nerve-centres.

The differences between cerebral and meningeal diseases in the human subject are arranged as follows by Aitken:—

Cerebral Disease.	Meningeal Disease.
1. From the outset, or from a very early stage of development, there is loss of some one or more of the proper nervous functions, such as paralysis, anæsthesia, loss of memory.	1. It is not till some time after the detection of signs of disease that diminution or loss of nervous function takes place.
2. Cerebral disease is not commonly attended by high-marked exaggeration of function, such as furious delirium, convulsions, intense hyperæsthesia or tenderness.	2. The subsequent diminution or loss of nervous function, which succeeds the prolonged existence of 'head symptoms,' is generally preceded in cases of meningeal disease by extremely severe excitement or exaggeration of functions, such as pain, tenderness, furious delirium, or convulsions.
3. Little vascular excitement attends cerebral disease, nor is there frequently any highly-marked general disturbance.	3. In meningeal affections there is usually much local vascular excitement, with general disturbance.
4. Paralysis and anæsthesia, losses of volition, ideation, perception, and the like, characterize cerebral disease.	4. Spasms or convulsions, pain and delirium, are the general features of meningeal disease.

Morbid Anatomy.—When the membranes of the brain are involved in the inflammation, the pia mater is thickened, opaque, and much congested over a larger or smaller area. The dura mater may be adherent in parts to the cranial bones.

Inflammation of the brain itself is usually limited to one or more patches, and, as in man, is more commonly met with as the result of injury or disease of the cranial bones. The inflamed brain-substance is sometimes much altered, but may be at first red and congested, afterwards becoming pulpy and disorganized, with congestion and œdema of the surrounding nerve-tissue.

The inflammation may subside, or terminate in suppuration.

The abscess when formed is generally single, but there may be several in pyæmia.

In a case recently before our notice, which was ushered in by violent frenzy, followed shortly by great depression, coma, and death, a large abscess was found occupying a large area of the right cerebral hemisphere.

Treatment—Bleeding is indicated in inflammation of the brain and its membranes when the fever is high and the excitement and restlessness very marked.

In cases of inflammation of the structures occurring in the course of a specific fever, or in some primary constitutional disease, bleeding is generally harmful.

In all cases, a cathartic should be given at once, as soon as the case is seen.

Locally, ice, or cloths steeped in cold water or some evaporating lotion, should be applied to the head during the stage of excitement. The animal should be removed from all noises, and kept as strictly quiet as is possible.

Internally, liquor ammoniæ acetatis and potassium bicarbonate may be given three times daily.

In the stage of irritation, potassium bromide or chloral hydrate may be given in addition.

These remedies may be given in the water, if the animal continues to drink.

The diet should be light and nutritious.

If paralysis and impaired sensation still continue after the abatement of the acute symptoms, a smart blister may be applied to the poll, and should be repeated if necessary.

CHRONIC DISEASES OF THE BRAIN.

CHRONIC MENINGITIS.—Chronic inflammatory changes are sometimes met with in horses in the membranes of the brain, but during life they are not usually diagnosed. They may give rise to general dulness, a staggering gait, slow pulse, and other symptoms, as well as sometimes to the more active manifestations of cerebral disturbance.

Chronic meningeal inflammation is usually the sequel of acute attacks of inflammation, or it may follow recovery from various injuries to the brain.

CEREBRAL SOFTENING.—Cerebral softening is usually the result of embolism of the cerebral arteries, but it may also follow inflammation of the brain substance. It sometimes occurs as a result of plugging of the cerebral arteries in pink eye or pneumo-enteritis.

The symptoms manifested during life, though not diagnostic, are similar to those met with in tumours and chronic meningeal disease of the brain, and were spoken of by the older writers as 'coma,' or 'sleepy staggers.'

If the softened patch of the brain substance be examined, it will be found variously altered.

It will be white, yellow, or pinkish in colour, and of variable consistency.

Softening, due to arterial plugging and atheroma, is usually met with in old animals.

HYDROCEPHALUS. — Hydrocephalus (ὑδωρ, water; κεφαλη, the head), or effusion of fluid into the ventricles of the brain alone, or into or beneath the arachnoid in addition, is of not uncommon occurrence in the horse.

In rare instances it is said to be met with sometimes in old animals, but is more frequently met with as a congenital defect in foals.

Its pathology is obscure, but when occurring in adults, it is probably, as in man, dependent upon pressure on the venæ galeni (D. Gresswell).

When occurring as a congenital defect, it is probably due to some constitutional cachexia in one or both parents.

Symptoms.—The symptoms of hydrocephalus may be arranged under two groups :—

1. *The Appearance of the Head.*—The hydrocephalic head is recognised by the great enlargement of the volume of the cranium.

The face appears very small by contrast, and the fontanelles and sutures are wider than in the normal condition.

Dr. Fleming, in his excellent 'Text-Book of Veterinary Obstetrics,' gives the following description of this unsightly condition :

'In some cases the distension has been so slight that the frontal bones are not much raised, while in others the collection of serum has been so great that these and other bones of the cranium are displaced, and the forehead, rising almost at right angles to the face, suddenly reaches an extraordinary height, giving the creature a startling appearance. The hydrocephalic tumour varies in figure as well as in volume. It is sometimes quite globular, and protrudes so high and so much over the face as to give the physiognomy a strangely human appearance ; in rare cases it is narrow, but excessively protuberant, involving only a part of the cranium ; at other times it is bilocular, and the divisions may be either alike or unequal in volume.

'Not unfrequently the diameter of such a tumour in the calf measures more than half a foot. The tumour is soft and depressible in parts, hard and resisting in others, owing to the bones of the cranium being altered and separated in places.

'These bones, and particularly the frontal, temporal and

parietal, are, as a rule, considerably deformed and thrown out of their natural direction, and in places so expanded and rarefied as to be no thicker than tissue paper. When the internal distension has been very considerable, their borders do not meet as in their normal condition, but are often widely separated, leaving between them vast fontanelles, occupied only by a thin translucent membrane, the dura mater, which is in immediate contact with, and closely adheres to, the skin.

'In some instances, especially in the calf, the bones on their upper part do not join at all, and the roof of the cranium, or rather of the cranial tumour, is entirely absent. In other instances, and particularly in the foal, a kind of bony arch extends from the nasal to the occipital bones, in the direction of the sagittal suture, with only here and there, on each side, small osseous patches from the parietal or temporal bones, and adhering to the dura mater.

'The tumour is always entirely covered by intact, though sometimes very thin skin, to which the hair is ordinarily attached, and is, indeed, at times longer than usual, especially at the sides.

'Owing to the great development of the forehead, the upper jaw appears to be shorter than usual; and, indeed, it will be found that it is really so.'

2. *General Symptoms.*—In the early stages the animal is irritable, and manifests febrile symptoms. Afterwards the foal becomes weak and debilitated, and its sensibility becomes impaired. Paralysis, coma, and convulsions precede death in fatal cases.

When hydrocephalus comes on in adult life, the symptoms are occult; but fortunately this disease is of rare occurrence, except as a congenital defect.

Prognosis.—Cases of recovery from congenital hydro-

cephalus are very rare, even in the mildest forms of the disease.

Morbid Anatomy.—The ventricles are found distended with a varying quantity of clear limpid or slightly yellow or greenish serum. The lining membrane of the sac is often rough and granular. The quantity of fluid varies very con-

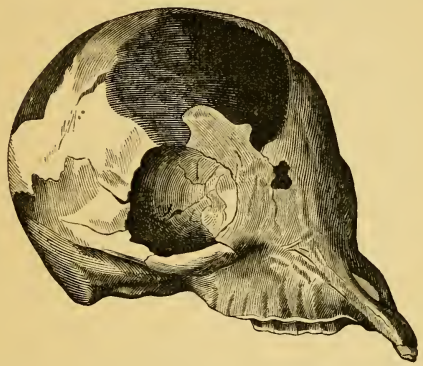


Fig. 4.—Head of Hydrocephalic Foal (from Fleming's 'Veterinary Obstetrics.'

siderably, but is generally from two to four pints in the foal and calf.

The late Mr. D. Gresswell collected as much as five and a half pints in one instance, and over eleven in another.

The largest amount recorded is two and a half gallons.

The accumulation of the fluid within the ventricles flattens the cerebral convolutions, and may almost entirely obliterate them, and the hemispheres are more or less completely destroyed.

The spinal cord, choroid plexuses, and cerebellum are not generally affected.

Treatment.—Even in the most favourable instances there is little profit to be derived from keeping hydrocephalic foals, as they are mostly very weakly, badly developed, and do not thrive well.

TUMOURS IN CONNECTION WITH THE CEREBRAL STRUCTURES.

TUMOURS IN CONNECTION WITH THE CRANIAL WALLS.

EXOSTOSES.—Osseous tumours, or ivory osteomata, have sometimes been met with growing from the internal, and also from the external, plates of the cranial bones of horses. They generally grow from the internal surface of the temporal bones, and are rounded, nodulated, and sometimes pedunculated.

They are hard and dense, being composed of compact ivory-like tissue, containing osseous corpuscles, but no Haversian canals or blood-vessels.

In some cases they become detached from the cranial walls, and lie loose in the cranial cavity.

TUMOURS IN CONNECTION WITH THE BRAIN AND ITS MENINGES.

1. TUMOURS OF THE CHOROID PLEXUS.—Abnormal growths in connection with the choroid plexus of the lateral ventricles are the most frequently encountered of all cerebral tumours in the horse.

They sometimes involve one of the plexuses, sometimes both, and are frequently symmetrical on both sides of the brain, not only in position, but in size also.

These tumours develop very slowly, and do not occasion

severe symptoms until they have attained a size perhaps as large as a pigeon's egg. They are sometimes met with as large as a hen's egg. They occur in horses of various ages, but are more common in old animals. The tumours are composed of cholesterine* ($C_{26}H_{44}O$), of calcareous particles termed 'brain sand,' and a little serum. They are enveloped in the folds of the choroid plexus, encapsuled by a more or less dense membrane, and held together in many instances by fibrous tissue in variable amount.

These tumours are developed in connection with hypertrophied villi of the choroid plexus, which are frequently enlarged and congested in old horses. Sometimes they are made up almost entirely of cholesterine arranged in spherical masses, and are probably due to caseous and calcareous degeneration of inflammatory exudation thrown out by the processes of the plexus.

The calcareous particles are contained in concentric bodies called corpora amylacea, and are termed 'brain sand.'

These concentric bodies, held together by fibrous tissue, are sometimes met with in the brain and its membranes in man, and are termed 'psammomata' (from ψαμμα, sand). In some instances these calcareous bodies form a large part of the tumours in horses, while in others cholesterine is the chief constituent.

The lateral ventricles may be almost, or quite, filled by the tumours, and not uncommonly congestion and other signs

* Cholesterine, a normal constituent of the bile and various tissues, especially the nervous, is a neutral substance which readily crystallizes from an ethereal solution in rhombic plates, which, when seen in mass, have a mother-of-pearl lustre. It fuses at 145°, is insoluble in water but soluble in alcohol, ether, and chloroform. When evaporated with nitric acid, the residue on addition of ammonia acquires a dull red colour. If sulphuric acid be added to an equal amount of solution of cholesterine in ether, the solution becomes red, then purple, while the subjacent layers acquire a distinct green fluorescence. Cholesterine is the chief constituent of gall-stones, and occurs in several other pathological products.

of recent inflammation of the neighbouring parts may be observed after death.

2. **CANCER.**—Cancerous growths are said to have been met with in rare instances in connection with the meninges and substance of the brain. When occurring here, these growths are nearly always due to secondary infection from primary cancerous disease of other organs of the body.

3. **MELANOSIS.**—Melanotic growths are sometimes observed in connection with the meninges and brain substance of the horse. Mr. Williams mentions a case in which black melanotic nodules were observed in the brain and membranes of an aged grey stallion which had suffered for some time from stringhalt. These nodules were also found along the spinal cord, and were especially numerous in the lumbar region.

4. **GLIOMA.**—Glioma is a variety of round-celled sarcoma, growing from the neuroglia of nerve-tissue. It consists of small round cells, and has been met with by Mr. Robertson on several occasions in connection with the cerebellum as an ill-defined tumour.

We have met with some patches of firm dense greyish tissue in connection with the cerebellum, but on examination they proved to be merely patches of sclerosis, no cells of a sarcomatous nature being found.

5. **FIBROMA.**—Fibromata have been sometimes observed in connection with the meninges of the brain.

6. **PARASITIC GROWTHS** are sometimes met with in connection with cerebral structures (vide Chapter on 'Entozoa).'

SYMPTOMS OF TUMOURS IN CONNECTION WITH THE BRAIN AND ITS MENINGES.

Tumours of the choroid plexus scarcely ever affect the health of the animal or give any signs of their presence until they have reached a size perhaps as large as a walnut or hen's egg.

When they have attained such a size, they may give rise at varying intervals to severe convulsive fits, staggering gait, and inability to perform work. In other instances no symptoms are manifest before a fatal stroke resembling apoplexy from rupture of a cerebral artery suddenly occurs. Sometimes the animal becomes violently excited, raises its head, and dashes violently forward. In these instances the head is hot, the pulse is full and bounding, and the vision is imperfect. After a while the paroxysm may be relieved, and the animal rises and regains consciousness.

When symptoms are manifested prior to the sudden attacks, there may, in some instances, be unconsciousness, with elevation of the head and fore-part of the body, while in others there is a tendency to keep the head depressed (Williams).

These tumours generally grow for a long time without producing any untoward symptoms, which mostly come on quite suddenly when the tumour reaches the size of a large egg.

All intra-cranial tumours may give rise to staggering gait, a tendency to fall asleep and become drowsy while eating, slow pulse and deep respiration—an aggregation of symptoms spoken of by the old writers as '*sleepy staggers*,' or '*coma*.'

Violent exertion is always liable to be attended with symptoms of cerebral disturbance indicated by the above manifestations, or by more active signs, as uncontrollable

excitement and frenzy. In some instances abnormal growths in the brain are also injurious in virtue of their being part of a general disease of the system, as, for example, in the case of melanosis or other malignant growths.

TREATMENT OF TUMOURS IN CONNECTION WITH THE BRAIN AND ITS MENINGES.

Drugs are of no avail in the treatment of these tumours, but careful management of the work and dietetic arrangements is in many instances attended with marked benefit.

DISEASES DEPENDING UPON ALTERATIONS IN THE CEREBRAL CIRCULATION.

MEGRIMS, OR CEREBRAL HYPERÆMIA.

We have already alluded to 'megrims,' or cerebral congestion, as being spoken of with other cerebral disturbances of an organic or functional character, as 'staggers' or 'vertigo.'

Definition.—By megrims we understand that form of cerebral disturbance usually sudden in onset, of short duration, liable to recurrence, and characterized by excitement, perversion or impairment of special sensation, but also to some extent of common sensation and voluntary motion (Robertson).

This form of cerebral disturbance is liable to be confounded with organic disease of the brain.

Etiology.—Cerebral congestions may be mechanical (or passive) or active.

Passive congestion is generally due to interference with the escape of the blood from the cranial vessels.

Active congestion may be due to increased cardiac activity or to disturbance of the vaso-motor influence, or may be

associated with general plethora, or with any interference with the general arterial or capillary circulation.

Megrims occurring in harness or draught-horses, except when due to organic cerebral disease, is almost always due to mechanical impediment to the flow of the blood from the brain, occasioned by the pressure of too tightly or badly fitting harness.

By some megrims is believed to be due to inflammatory action, but there does not appear to be any grounds for this supposition.

It is said also that megrims may be produced by exposure to the rays of the sun, or by driving fast after a heavy meal.

These causes certainly may increase the tendency to this affection, but it is very improbable that they alone can cause it. As already pointed out, however, indigestion is liable to be caused by fast driving after heavy meals, and may induce vertiginous symptoms, which might easily be confused with megrims depending upon actual congestion.

Symptoms.—There are generally no premonitory symptoms, the attack being sudden in its onset.

The animal slackens its speed, or stops suddenly, and moves its head from side to side or up and down. Sometimes its head is turned to one side.

The vessels of the face and throat are engorged, the eyes stare, the nostrils are dilated, and the breathing is rapid. The skin may be bedewed with perspiration, and the cervical and facial muscles twitch convulsively.

If the collar causing the obstruction be removed, the symptoms abate, and the animal soon recovers.

When the symptoms are very severe, there is great excitement, the convulsions become still more severe, and the animal falls prostrate to the ground.

Morbid Anatomy. — The brain is found congested,

and the venous sinuses and vessels are engorged. The capillaries of the pia mater are full of blood, and the brain-substance, when cut into, presents a large number of red spots, and is redder than natural.

Treatment.—First remove the collar to permit of the return of the blood to the heart, and then apply cold water to the head.

When the affection depends upon gastric disturbance, this must be treated as already directed (*vide* 'Diseases of the Stomach').

In those instances where the neck is peculiarly shaped, it may be necessary to use a breast-strap instead of a collar.

CEREBRAL ANÆMIA.—Anæmia of the brain of the horse may depend upon general anæmia, feeble action of the heart, or upon local interference with the blood-supply from intra-cranial obstruction. The brain and its membranes become paler than natural, and occlusion of some of the small vessels may be observed. When dependent on general anæmia, the symptoms are early exhaustion, general pallor of the visible mucous membranes, and dilated pupils.

In partial anæmia the symptoms vary in accordance with the nature of the cause.

The Treatment of anæmia of the brain, when due to general anæmia, consists in attention to the hygienic conditions, good nutritious diet, and the administration of iron and vegetable tonics.

CEREBRAL EMBOLISM AND THROMBOSIS.

Cerebral embolism denotes a plugging of one of the arteries of the brain by a small vegetation or fragment of fibrin detached from the heart or aorta, and swept off into

the current of the circulation. The embolism may also be due to detachment of fragments of pulmonary thrombi.

Cerebral thrombosis is almost always associated with an unhealthy condition of the inner coat of the vessels, or to a diseased condition of the blood itself.

Embolism and thrombosis cut off the supply of blood from the part of the brain to which the diseased vessel is distributed, and cerebral softening ensues.

CEREBRAL HÆMORRHAGE, OR APOPLEXY.

Cerebral hæmorrhage is due to rupture of a vessel in the brain. Rupture of cerebral arteries is generally due to disease of the walls or to changes in the surrounding parts by which the vessels are deprived of support, or finally to injury. Cerebral hæmorrhage is very rarely met with in the horse.

Symptoms.—In some instances there may be premonitory symptoms, but generally the horse suddenly loses consciousness and falls prostrate to the ground.

The extravasation, if occurring on one side of the brain, gives rise to partial or complete hemiplegia, or a certain set of muscles, or one limb only (monoplegia) may be paralysed, in accordance with the extent of destruction of the cerebral substance. The symptoms vary in accordance with the part of the brain affected, and in rare instances, owing to increasing hæmorrhage, may take some days before they are fully developed.

The breathing is stertorous and the pupils are usually dilated and insensitive to light, and the animal is insensible to external surroundings.

The pulse is rapid and thready ; the limbs are flaccid, and the body is bedewed with a cold sweat.

When the lesion is very extensive, the coma is profound and the sphincters relaxed.

Prognosis.—When the hæmorrhage is extensive, it generally proves fatal. In less severe cases recovery may take place, but more or less impairment of voluntary motion may remain.

Morbid Anatomy.—Cerebral hæmorrhage in the horse occurs in the substance of the brain, but it may also occur in the meninges, especially when due to injury. When the hæmorrhage is recent, a red clot of variable size is found. This afterwards becomes encysted, and undergoes changes in colour and consistence, and hæmatoidin crystals and granular pigment are often found in it.

In other instances inflammatory changes, resulting in the softening of the cerebral structures, or abscess may be formed.

In old horses, the cerebral arteries will probably be found to be in an atheromatous condition.

Treatment.—In those instances of cerebral hæmorrhage where the coma is attended by full strong cardiac action in the young and previously healthy animal, bleeding is recommended.

In other cases, all that can be done is to apply cold to the head, which should be elevated a little to allow a good supply of fresh air, and to bandage the legs. If collapse should threaten, owing to intensity of the shock, stimulants may be administered through enemata. In all cases the bowels should be freely acted upon.

Section II.

DISEASES OF THE SPINAL CORD.

INFLAMMATION OF THE SPINAL CORD AND ITS MEMBRANES.—Inflammation of the spinal cord (myelitis), and of its coverings (spinal meningitis), may be described together, as in many instances both the cord and

its membranes are simultaneously involved, and where the affection is mainly confined to one, it is often difficult to diagnose which of these structures is the chief seat of inflammation.

Etiology.—Spinal inflammation may be acute or chronic. Acute spinal inflammation may be due to injury, such as fracture or displacement of the vertebral segments resulting in compression of the cord.

When arising idiopathically, it is often very difficult to trace the cause, but hereditary predisposition is certainly in many instances traceable.

Exposure to cold and damp, especially after a heavy day's work, are potent causes of inflammation of the spinal cord, and of its membranes.

Symptoms.—Premonitory symptoms are more likely to be manifested when the membranes are the special seat of inflammation, whereas they are more likely to be absent when the inflammation is especially concentrated in the cord itself.

Before paralysis comes on, in cases of inflammation of membranes especially, spasmodic contractions of the large muscles connected with the trunk and limbs, chiefly the hind ones, are observed.

Febrile symptoms are manifested, and perspiration bedews the body. The legs are lifted up and placed upon the ground in quick succession. The animal suffers great pain, and is very restless and excitable, and is disturbed by the slightest movements of the attendants.

Now and again he may have intervals of calm and quiet, and cessation from spasms.

But after the lapse of eight to twelve hours or so, from the pain caused by progress of the inflammatory action, and from the exhaustion, the animal falls prostrate to the ground.

The spasms often continue as he lies, and in very severe cases gradually increase in frequency and intensity.

Sensation is much impaired in the limbs; the pulse is quick, and the artery full and firm; the respirations are accelerated, and the temperature is elevated.

The appetite is impaired, or lost altogether; the urine is more scant, and the bowels are usually confined.

In those instances where the inflammation is more probably seated in the cord itself specially, the mode of onset differs from the above.

No symptoms of spinal inflammation are observed in these cases until sudden interference with the power of locomotion attracts the notice of the attendants. The loss of motor power becomes in a very short time so complete that the animal is prostrate, and is unable to rise.

The muscular spasms, so prominent a feature in cases where the meninges are especially involved, are not so marked or so frequent in these cases.

Sensation is rarely, if ever, entirely destroyed, pain being felt when a limb is pricked. Pain, when severe, is associated, as indeed is severe pain of other organs, with acceleration of the pulse and respirations.

Symptoms of Chronic Spinal Inflammation. — The animal has tonic and clonic contraction of certain muscles, or sets of muscles.

The spasms often gradually increase in frequency and, indeed, in intensity, but are rarely of such a severe description as to prevent the animal from working.

They may afterwards subside, or pain and increased loss of power over certain sets of muscles may ensue, as well as rigidity or spastic contraction in different groups of muscles. The animal does not show any tendency to lie down, and, when down, has great difficulty in rising.

These symptoms slowly and gradually progress, and the

loss of muscular power is often followed by complete paralysis of the posterior extremities.

In those instances of chronic inflammation of the spinal cord specially, as distinguished from that of the meninges, loss of motor power is mostly followed by more or less complete paraplegia.

Morbid Anatomy.—After death from acute spinal inflammation, the cord and its membranes are more or less congested, sometimes, indeed, very highly. The sub-arachnoid space is filled with serous exudation, and the cord, when cut into, is much redder than normal. The post-mortem changes, however, are sometimes very slight in comparison with the severity of the symptoms manifested during life, and they vary greatly in extent and in character.

In some instances the cord becomes soft and pulpy, the nerve-fibrils and cells undergo marked alterations, which are more especially, however, concentrated in the grey matter.

In chronic inflammation, the cord is red, injected, and softened; the meninges are thickened, and the arachnoid is covered with inflammatory exudation, by which the two surfaces may become united, or the cavity is filled with reddish serum.

Diagnosis.—The history will in many cases help to clear up any doubts as to the diagnosis of the case. Acute spinal inflammation may be confused with azoturia. The dark colour of the urine in the latter affection, however, is not a feature of spinal inflammation, and the spasms are mostly limited to the gluteal region. In acute spinal inflammation a greater number and variety of muscles are usually affected, and the spasmodic contractions are of a more clonic nature.

Broken back is generally directly traceable to an injury,

whereas spinal inflammation is mostly of idiopathic origin.

Prognosis.—Acute spinal inflammation usually terminates fatally.

Chronic inflammation does not generally end fatally, but so much loss of power remains in many instances that treatment is often abandoned.

Treatment.—A purgative, such as a full dose of aloes, should be administered at the outset, and the bowels should be kept carefully open during the progress of the disease by enemas, or the administration of saline purgatives.

Bleeding may be in many instances adopted, but it is contra-indicated when paralysis has set in.

Cloths wrung from hot water medicated by the addition of belladonna may be applied over the region of the spine to ease the pain and subdue the inflammatory action.

Internally, belladonna, in the form of extract or tincture, may be given three times daily.

After the acute stage is passed, ergot of rye, in the form of the ethereal tincture, and potassium iodide, may be tried, and where deficiency of nerve-power is left, stimulants may also be administered.

Externally, if impairment of nerve power remains, some authors recommend the application of the actual cautery over a considerable length of the spine, on both sides.

If blisters be used, cantharides should not be the active ingredient.

Internally, after the acute stages are over, strychnine and quinine may be administered three times a day.

In those cases where micturition is not easily performed, the catheter should be passed twice or three times daily, in order to draw off the urine.

The food should be good and nutritious, and the general hygienic conditions should be attended to.

SPINAL HÆMORRHAGE.

In rare instances, hæmorrhages in connection with the spinal cord have been met with from a diseased condition of the vessels. Generally, however, hæmorrhage is a surgical lesion, the result of direct violence.

The cord itself may be the seat of the hæmorrhage, or this may occur in the sac between the ensheathing meninges, or finally it may occur outside of the membrane.

If the extravasation into the substance of the cord be large, paraplegia may suddenly come on in consequence, but when it is of less extent, exaltation of functional activity may or may not be followed by impairment or loss of motor power.

TUMOURS AND ADVENTITIOUS GROWTHS IN CONNECTION WITH THE CORD.

Exostoses, melanotic growths and cancerous growths, are sometimes met with in connection with the cord.

We have already alluded to a case of melanotic deposits in the cord.

An account of a very interesting case of cancer of the spine is thus recorded by Mr. Hunting :

On the 10th September, 1855, I was requested to see a chestnut mare. the property of Mr. G. S. R., of S. H. She had been unwell for ten or twelve days with cough and sore throat ; her neck was likewise very stiff, but her appetite had remained good up to yesterday, when she became tympanitic, and suffered intense pain. When I saw my patient, I found that the pulse numbered 68, and was weak. The submaxillary artery appeared full and soft, and the action of the heart feeble. The breathing 59 in the minute, and rather laboured. The conjunctival membranes were very much injected, and of a yellowish colour. The mouth was hot and dry. The nostrils were greatly dilated. The ears were cold, legs warm. The fæces were of a healthy character. The respiratory murmur throughout the whole length of the trachea was much louder than in health, and the left lung gave evidence of partial congestion. The cough was thick and heavy, and of a peculiar sound, but not frequent. The larynx and trachea were very painful on pressure.

A watery fluid flowed from the eyes, but there was no discharge from the nostrils. The neck was very stiff, so much so that the animal could neither eat nor drink from off the ground, nor move the head in a lateral direction. The parotid glands were much larger and harder than usual. The appetite, however, was but little impaired.

From these symptoms I considered it to be a case of ordinary influenza, or distemper, which disease was exceedingly prevalent in the neighbourhood at the time. Acting upon this impression, I treated it as such until the 24th, when my patient was so much better, that professional attendance was no longer necessary. The head could now be moved with greater freedom in a lateral direction, and the mare was enabled to eat and drink from the ground. The breathing had become natural, both in character and frequency. The pulse 38, and healthy in tone, the appetite good, the animal lively, and capable of taking half-an-hour's exercise daily.

On the 6th of October I received a message to say that the mare was not so well. On arriving at the place late in the day, I found her apparently suffering but little pain, the intense agony which the owner had observed in the morning having passed off. The other symptoms they described as also existing were an enormously distended abdomen, frequent groaning, rigid limbs, an occasional lying down but quickly rising again, very heavy breathing, and an anxious expression of the eyes.

The pulse was 44 in number, and rather weak at the jaw; the sounds of the heart were so feeble that they were scarcely audible on the left side; the breathing was 66 in the minute, but not laboured; the motion of the abdominal muscles was indeed so slight, that I was unable to take the number of respirations at the flank; the neck was still a little stiff; the nostrils dilated, and much anxiety of the countenance present. The mucous membranes were healthy in colour; the mouth cool and moist; the surface of the body of a natural temperature; the bowels regular, and the urine of a light colour.

On the application of pressure to any part of the spinal region, from about the tenth dorsal vertebra to the sacrum, the whole of the voluntary muscles behind became as rigid and as hard as in the worst cases of tetanus during the periods of excitement. Very little difference could be detected in the violence of muscular contractions, whether the pressure was employed directly over the spine, or within twelve inches on either side of it. This tetanic rigidity sometimes occurred when pressure was not applied, and also when the animal was made to back, but then in a much less degree, and lasting for a few minutes only.

On an examination per rectum, I detected a large tumour on the left side of the spine, having a density, as imparted to the feel, equal to the structure of the liver. It appeared to be about eight or nine inches in diameter, three inches thick in its centre portion, and an inch at its circumference. It was closely connected to the posterior part of the kidney, over-lapping the posterior aorta, and extending to the right side of the spine. On pressing the enlargement, evident pain was obtained, but the tetanic spasm did not follow, nor was there the slightest indication of pain when pressure was applied to the inferior portion of the lumbar vertebræ. The pulsation of the

posterior aorta between the tumour and the bifurcation of the vessel into the iliac arteries was scarcely to be felt, which I attributed to pressure of the enlarged mass upon the aorta.

I looked upon the case as one of very doubtful recovery, thinking that I had most likely to deal with the formation of an internal abscess as a sequela of influenza, which is not a very uncommon occurrence ; but my patient's appetite was good, the heart's action not much disturbed, and the fæces and urine healthy. I considered treatment justifiable. Counter-irritants were therefore applied to the loins, and pot. iodidum given internally with vegetable tonics. Under this treatment the tumour gradually became less in size, and much softer in consistence. The rigidity of the muscles was likewise less violent when pressure was applied to the spine.

On the 24th a 'charge' was applied to the whole of the lumbar region ; mineral and vegetable tonics were daily given with the iodide of potassium, and exercise was enjoined.

From this date up to the 18th of November there was a gradual wasting away of the muscles; the appetite was generally good, the animal eating an average quantity of the most nutritious provender that could be obtained.

During this period the urine became highly impregnated with albumen, but which gradually diminished in quantity, until on the 18th of November, after which time I entirely failed to detect its presence.

On several occasions during the above period, the peculiar spasmodic contraction of the muscles, the tympanitic state of the abdomen, the anxious and protruding eyes, and the intense suffering would come on, but lasted only for a short time. These symptoms generally occurred two or three days in succession, and were not again seen for six or eight days.

On the 18th of November I found that the tumour was very much smaller, and also soft and flaccid. The pulse and breathing were perfectly natural, both in character and number. The spasmodic contraction of the muscles did not occur even when pressure was applied to the spine, and the animal looked more cheerful and lively.

From this date up to the beginning of December the mare slightly improved in condition, and I had more hopes of her ultimate recovery, but on the 6th of December the symptoms again returned, and in as bad a form as before. They continued for several days, and then disappeared.

On the 23rd of December, at eight o'clock in the evening, she was left apparently no worse. She drank an unusual quantity of water, and ate her food with avidity ; but early on the following morning she was found dead and cold.

Post-Mortem Appearances.—The thoracic viscera, the spleen and the liver, were all very pale in colour, but otherwise healthy. The left kidney was very much enlarged. The stomach, intestines, bladder, uterus, and right kidney were likewise healthy ; but the pancreas was filled with small tumours, varying in size from a pea to a walnut, which contained a yellowish jelly-like substance. The spinous processes of the lumbar vertebræ were extensively diseased. The second, third and fourth cervical vertebræ were extensively diseased. In the broad, flat spinous process of the dentata, there existed a circular aperture, extend-

ing from side to side, and from the arch to the top of the process at the point of its bifurcation. On the lateral and anterior part of the body on the left side the disease had made equal ravages, extending into the foramen at the base of the odontoid process. Immediately above the transverse process, on the same side, the destruction of bone extended from an inch and a half upwards and backwards, invading a nearly circular portion, and removing a large part of the articulation formed by the left half of the spinous process of the dentata and the anterior articular process in the third vertebra. At the antero-inferior part of the body of the bone, the disease had established a complete communication, nearly an inch in diameter, with the spinal canal, but the dura mater was not destroyed.

In all the affected portions there was a considerable quantity of bony material removed, forming large cavities, which were filled with a reddish-looking mass, presenting precisely the same appearances as those of the lumbar spine. In the third and fourth vertebræ the lesions were less extensive, but showed the same characteristic appearances.

Symptoms of Tumours of the Cord. — The symptoms of spinal tumours will necessarily vary much, in accordance with the seat and extent of the tissue invaded.

In many cases there is muscular spasm, followed afterwards by loss of motor power. There is pain on pressure over the seat of the tumours, and symptoms are of slow progress in most instances.

In those cases where symptoms of paralysis or loss of function are manifested, recovery cannot be hoped for, and treatment is of little avail.

SCLEROSIS OF THE NERVE-CENTRES.

By sclerosis of the nerve-tissue we understand an induration or hardening of the textures in varying extent and situation.

Sclerosis of the nerve-centres is not of common occurrence among horses. We have met with more than one instance of sclerosis affecting the cerebellum.

Mr. Robertson also records some instances affecting the cerebellum and the spinal cord in adult horses which had been subjected to hard work.

Etiology.—Hereditary predisposition, overwork, exhaus-

tion, are among the chief causes of sclerosis. It has been observed that the families of animals so affected were subject in some instances to various nervous affections, such as spasms, which were generally of a choreic nature. Sclerosis may also follow as a sequel of some affections, such as certain fevers or inflammatory actions involving various parts of the nervous system.

Symptoms.—When the sclerosis involved the lobes of the cerebellum, it was noticed that the animal showed a tendency to execute gyratory movements, and had spasmodic contractions of the muscles of the eyeballs.

In all scleroses in the first instance, the motor power appeared to be more affected than the sensory.

Morbid Anatomy.—Pale greyish patches of varying extent may be found at the seat of disease. These changes have been observed especially on the lateral lobes of the cerebellum. In one case under our notice these changes were confined [to the left lobe of the cerebellum; the cerebral hemispheres were healthy.

Sclerosis of the nerve-tissue is characterized by the growth of a large quantity of fibrous tissue, and by atrophy and degeneration of the nerve-elements themselves.

The tissue resembles such as is formed in productive inflammation, and sclerosis has in consequence been regarded as of inflammatory origin.

Some authorities, however, view the increase of connective-tissue as due to hyperplasia of the neuroglia, the cause being unknown (Green).

Treatment.—Nux vomica, or its alkaloid strychnine, with other vegetable tonics and iron, in conjunction with a good nutritious diet, are likely to prove beneficial, though complete recovery is perhaps only to be expected in very exceptional instances, where the nervous changes have not made any great progress.

CHOREA.

Definition.—Chorea is a peculiar disorder characterized by irregular clonic contractions of different muscles, especially those of the posterior extremities.

Nature and Etiology.—There is great doubt as to the true nature of chorea. As in man, it is of more frequent occurrence in the young, but though similar in many points in the equine species, it nevertheless cannot be regarded as strictly analogous with chorea in man.

Hereditary predisposition is perhaps the chief cause of chorea, though malhygienic conditions, overwork and exhaustion, may also act as exciting causes.*

In the horse, chorea is only exceptionally the sequel of other diseases, such, for example, as the specific fevers.

Several theories have been propounded from time to time to account for chorea.

By some it is regarded as a purely functional disorder, *i.e.*, unassociated with any organic change.

By others it is held to be due to some diseased condition of the blood, probably associated with rheumatism.

In some instances, at any rate, organic changes have been found after death.

Stringhalt, an involuntary convulsive action of the muscles of one or both hind-legs, occasionally affecting the fore-legs, may be regarded as a choreic affection.

Stringhalt probably depends upon some special lesion interfering with the functions of the nerves supplying the affected muscles, or of the spinal cord itself.

* Mr. Williams, in his 'Principles of Veterinary Medicine,' says he has on record one instance where four young horses, the progeny of a dam which was affected in the back, died from spinal paralysis before they had attained the age of thirteen years. A fifth showed signs of aggravated nervo-muscular disturbance.

Sometimes, however, it is of a reflex character, being due to peripheral irritation, as, for example, when a bone spavin presses upon the bones of the hock (Williams).

In grey horses, however, it has been occasionally observed to be due to a deposit of melanotic matter in the sheath of the great crural nerves, and two cases are recorded in which it was due to exostosis of the ilium pressing on the sciatic nerve.

Symptoms.—The spasms occurring in chorea are sometimes very persistent, at other times they only occur at long intervals.

They present every degree of intensity, sometimes being very slight, sometimes very severe.

'*Shivering*' is a peculiar nervo-muscular disorder affecting the muscles of the back and posterior extremities.

When a horse the subject of this affection is backed or turned, the muscles of this region are thrown into clonic spasm, contracting and relaxing irregularly. The tail is often spasmodically elevated, and then depressed. When the horse is trotted forwards, the spasmodic contractions are very seldom developed, but they may be brought into action by the head being turned round rapidly.

In some instances the spasmodic contractions of the muscles of the loins and thighs come on while the animal is drinking. Though the posterior is more frequently affected than other parts, choreic disturbance may also affect the muscles of the neck and anterior extremities.

The spasmodic contractions in this region are very variable in severity and in the time of recurrence.

Morbid Anatomy.—Though in many instances nervous lesions have been found in connection with the brain and cord in chorea, yet no one in particular can be said to be specially characteristic of this affection.

In some instances tumours have been found in the

ventricles. Melanotic disease was the cause of chorea in a case recorded by Professor Williams. In one instance before our notice, hyperæmia of the cord and of the basal ganglia of the brain was observed.

Effusion into the sub-arachnoid cavity of the brain and cord, with thickening of the serous covering of the brain, and various changes in the nerve texture, are also mentioned as having been met with.

Treatment.—In the first place it is necessary in all cases of chorea to remove the cause when that is possible.

The general hygienic conditions should be carefully attended to; the diet should be good and nutritious, and the work proportionate to the strength of the animal.

Internally, mineral tonics, such as salts of iron, zinc, or Fowler's solution, with vegetable tonics, are worthy of trial.

EPILEPSY.

Definition.—Epilepsy is a peculiar affection of the nervous system, characterized by sudden temporary loss of consciousness, and associated for the most part with a convulsive attack which, in many instances, cannot be traced to any organic disease.

The fits are of variable severity and duration, and may be succeeded by debility, and sometimes by a drowsy or semi-comatose condition.

Pathology and Morbid Anatomy.—There are not many affections of the pathology of which we are so much in doubt as we are about that of epilepsy.

With regard to the causes of this affection we know but little definitely, but hereditary predisposition is very probably, as in chorea, an important factor in its production.

Epileptic attacks, being manifested under very different

conditions of the system, cannot be regarded as a distinct and separate affection. Like jaundice, epilepsy is an accompaniment of several different diseased states.

In many cases after death no organic lesion is discoverable in epileptic animals, and the affection is in these instances regarded as functional.

It may depend upon disease of the brain, such, for example, as the deposition of morbid growths and inflammatory action, or other changes in the texture of the cerebral structure.

In some cases in man, epilepsy has been proved to be due to irritation of the brain caused by the projection of pieces of bone. This may also be a cause of this disease in the horse in some instances.

Finally, epilepsy may depend upon alterations in the blood-supply to the brain, and may be caused by reflex irritation from various distant organs in a state of disease.

The medulla oblongata and upper part of the cord are regarded by Dr. Reynolds as the primary seat of epilepsy. Some observers are of opinion that the parts primarily affected are the medulla oblongata, the corpora striata, and other parts of the base of the brain.

Brown Séquard has shown that epilepsy can be artificially produced in the guinea-pig by section of one of the lateral columns of the cord, anywhere between the medulla oblongata and the tenth dorsal vertebra. It must, however, be remembered that convulsion is by no means the most distinctive element in an epileptic attack. And, indeed, however seriously the cord and medulla and ganglia at the brain base may be affected subsequently in an attack, the earliest phenomena of an epileptic seizure must be connected with some limited spot in the nerve-centres, which, though different in different cases, is probably always the same for the same case (Bristow).

In some instances of post-mortem examination in the horse, extensive disease of the membranes has been observed; but in many cases no abnormal lesions whatever were discoverable.

Symptoms.—In the human being an epileptic attack is not unfrequently preceded by what is termed the 'aura,' which is a subjective sensation of coldness, heat, tingling, etc. This, though of course not detectible in the horse, may likewise precede the epileptic seizure.

The horse, when attacked, staggers, champs his jaws, becomes unconscious, and falls to the ground convulsed.

Sometimes the spasms are very slight, and the animal quickly regains consciousness and seems as well as ever.

Sometimes the spasms are confined to one limb, sometimes to one side of the body, or to the muscles of a particular region, as the facial and cervical region.

In some instances the convulsions are very severe, and the breathing seems to be stayed for a time by the tense contraction of the respiratory muscles.

The animal froths at the mouth, grates its teeth, moves its head quickly to and fro, and tosses about wildly.

Opisthotonos, or bending of the trunk backwards, is sometimes produced by spasmodic contraction of the muscles.

The pulse is generally full and accelerated, though it may be weak and not much above the normal in frequency.

After a while, when the tonic spasms, succeeded by the clonic spasms, have abated, the animal regains consciousness.

Sometimes he remains dazed, as it were, semi-comatose; at other times he may manifest great weakness.

Treatment.—During the paroxysm cold water may be dashed upon the head, and all means should be adopted to prevent the horse harming himself in his convulsions.

In plethoric animals bleeding is recommended by some authorities.

After the attack is over the general health should be attended to, the diet carefully regulated, and the bowels opened.

If the disease depend upon peripheral irritation, as, for example, that which may be set up by worms, anthelmintics and purgatives should be administered.

In other cases a course of bromides may be administered. Bromide of ammonium, potassium and sodium, combined with a vegetable tonic, are perhaps the most efficacious of all remedies in subduing epileptic attacks when traceable to disease of the medulla and brain.

Belladonna is also of value in the treatment of epilepsy, and may be tried should the bromides fail.

Other remedies which have been recommended in epilepsy are zinc, silver, iron, and arsenic salts.

TETANUS OR LOCK-JAW.

Definition.—Tetanus is a diseased condition characterized by tonic spasms of the voluntary muscles, and to some extent, probably, of the involuntary muscles also.

The spasms are painful, and from time to time undergo exacerbation, followed by intervals of repose.

Etiology and General Pathology.—Of the domestic animals, the horse and sheep are the most liable to tetanus; but the disease is very rarely seen in the ox tribe.

Tetanus frequently arises in connection with wounds, though it also occurs without any obvious cause whatever. When traceable to an injury, it is spoken of as traumatic tetanus; when it arises without apparent cause, it is called idiopathic tetanus.

The liability to traumatic tetanus is in no way dependent

upon the severity of the injury, as this affection may follow even very slight wounds. It is most likely to follow lacerated wounds or punctures.

Wounds in the feet, thighs, quarters, and forearm are especially liable to be followed by tetanus; and this is more particularly the case when the nerves are injured.

Wounds, it will thus be seen, in parts which are the most tense, and in structures bound together by strong unyielding tissues, are more frequently succeeded by tetanus than injuries in the laxer tissues.

Injuries in the joints, although frequently inducing a high febrile condition, are nevertheless not often followed by tetanus.

The operations after which the disease most commonly supervenes are docking and castration; and, in some instances, the insertion of setons has been followed by an attack.

In some cases tetanus has been traceable to irritation of the stomach and intestines, caused by worms or collections of sand in the large intestines.

Uterine irritation following abortion is also a cause of tetanus in some instances.

Exposure to cold and damp, especially after exhaustion, may induce tetanus.

Horses, when exposed in cold weather after being clipped, are sometimes affected, and sheep similarly, when exposed in cold and boisterously wet weather immediately after being shorn, are said to be very liable to become affected with this disease.

Tetanus is more prevalent in certain districts than in others. In some parts of the country it is very frequently met with, while in others it is as rarely seen.

Sometimes it occurs as an enzootic disease, and Mr. Williams records that in the summer of 1858 he witnessed

10 cases in a fortnight, of which some were traumatic, the others not being traceable to any injury.

In the human species tetanus is often idiopathic in hot countries.

Of the true nature of tetanus there is considerable doubt. Some hold that the traumatic variety is due to irritation of the nerves implicated in the wound, and that the muscular spasms result from reflex irritation.

In support of this it is argued that many cases of tetanus following docking have recovered after repeating the operation higher up. Yet it must be remembered that recovery by no means invariably follows the repetition of the operation, and, moreover, some cases of traumatic tetanus recover without any treatment whatever.

By some authorities tetanus is considered to be a blood disease, and several reasons have been advanced in support of this view, viz.,

(1.) Tetanus often occurs idiopathically without any injury.

(2.) It is more prevalent in certain districts than in others, and is sometimes enzootic, affecting several animals in the same district. There is, however, no proof that tetanus is ever contagious.

(3.) The resemblance to hydrophobia and strychnine poisoning suggests a similarity in the nature of the cause.

(4.) Tetanus is said to be transmissible to men by eating the flesh of animals which have died of this disease. Of the truth of this the herdsmen of the districts on the River Plate are confident; yet, nevertheless, confirmatory evidence seems to be lacking. Mr. Robertson records that he has known of dogs fed on horses which had died of tetanus, and does not recollect any evil results ensuing in consequence.

Other reasons have also been given in support of the view that tetanus is a blood disease.

The assertion that the spasms begin in muscles which have no anatomical connection with the injured nerves cannot be regarded as of any weight whatever in deciding in favour of the affection being a blood disease.

In what manner is the blood then altered?

Are we to look to changes in its quality, or to the presence in it of some endopathic poison resembling strychnine in its effects, as suggested by Sir T. Watson and Dr. Richardson? Or are we to look for the presence of living microbes in the blood, as, indeed, have been demonstrated by M. Pasteur in patients suffering from hydrophobia?

This question for the present remains unsettled, but it seems not improbable that tetanus may eventually prove to be due to the presence and development of living germs in the blood and tissues.

We may, perhaps, the more easily understand tetanus as a disease if we first consider cursorily some of the phenomena exhibited by a tetanized muscle.

If a nerve-muscle preparation be carefully made and preserved, and the nerve be irritated in any way whatever, the muscle will respond to the application of this new incident force by an aggregate of changes which are collectively spoken of as a contraction.

Further, if during the period of maximum intensity of the contraction another equal stimulus be applied, the additional response will be nearly equal to the first.

A lever suitably attached to the muscle will be raised very nearly as much again, the amount of the two contractions will be nearly twice that of the first alone. If more stimuli of equal intensity be successively applied, with each subsequent irritation, the amount of the consequent contraction becomes gradually less, until at length, when the stimuli reach a certain number in a given time, the lever is not raised higher, but the component contractions are apparently united, fused, as it were, to form a single continuous uniform contraction, and though really still existing, are not to be indicated by ordinary instruments.

This condition of a muscle, which is called tetanus, may be produced by the interrupted current, or by any rapidly repeated shocks. The frequency of the stimuli needed to produce tetanus differs in different cases.

'Kronecker and Stirling find that ten stimuli per second will cause complete tetanus in the red muscles of the rabbit, whereas the pale muscles require at least double that number.

'When the stimuli are repeated more frequently than is required to bring about a complete tetanus, the constituent contractions are still proportionately increased in frequency. This is shown by the increased pitch of the muscular sound.

'Kronecker and Stirling, by using a special instrument for rapid interruption, the so-called tone inductorium, have been able to obtain in all cases a complete tetanus with alternating induction shocks, even when repeated, they believe, as frequently as 22,000 times a second' (Foster).

Thus it appears that the number of constituent contractions which may coalesce to form a tetanus is very great indeed, and we can only conclude from this that in the disease where so many muscles are involved the expenditure of force entailed is enormous, even if we suppose that the spasms are not of a very complex character, that is, are not constituted by a large number of component contractions.

Every time a muscle contracts there is manifestly an expenditure of energy. Work is done; force is supplied, directly or indirectly, by means of the dissociation of highly complex organic compounds, of whose nature little is known, and thus waste products are produced.—*From an article in the* VETERINARIAN (*Oct.* 1882), *by Mr. J. B. Gresswell.*

Varieties and Symptoms.—There are several varieties of tetanus, which have received various names from the particular muscles mainly involved.

As a general term tetanus comprises all the varieties, and involves a greater or less number of the voluntary muscles, and probably also some sets of involuntary muscles.

When the muscles of mastication are alone or in chief part affected, the condition is called trismus, or lock-jaw. This is noticeable as being in most cases of tetanus a prelude to the succeeding symptoms.

When the superior cervical and dorsal muscles are especially affected, producing elevation of the head, and curving downwards of the spine, the affection is called opisthotonos.

When the muscles of one side are affected, the condition is called pleurosthotonos, or tetanus lateralis.

When the inferior muscles are involved, the chin drawn

towards the heart, and the spine arched backwards, the condition is spoken of as emprosthotonos.

This latter condition is very rarely met with in horses, but has been observed on one occasion at any rate, being produced by rigid contraction of the inferior muscles.

Generally, however, in the horse trismus with opisthotonos is the condition met with; pleurosthotonos is sometimes, though not frequently, observed.

Tetanus may be acute, sub-acute, or chronic.

When this affection owes its origin to a wound, it usually manifests itself in from ten to twenty-eight days after the infliction of the injury.

Generally there is first noticed a stiffness about the neck and lower jaw, and of the muscles near the seat of injury, and there is difficulty in mastication and deglutition, together with increase of the secretion of saliva, and a peculiar champing of the teeth.

If the head be suddenly elevated, or the horse suddenly disturbed, there is a characteristic protrusion of the membrana nictitans over the eyes, which are withdrawn within the orbits, thus causing the animal to show the white conjunctival surface at every convulsive retraction.

As the disease advances, the stiffness becomes more marked, especially in the muscles of mastication and in those of the upper part of the neck, and soon spreads to other groups of voluntary muscles, especially those of the abdomen, back, and hind-quarters.

At length the tetanic condition becomes fully established, and is very apparent even to a superficial observer.

The limbs are extended, and kept apart; the tail is elevated, and the animal moves in a peculiar, stiff, straddling way, with difficulty and pain.

The pulse is generally not much affected in the first in-

stance, but in a day or two it becomes accelerated, and the artery becomes firm, hard, and incompressible. In very acute cases, however, the pulse may become much accelerated in the very early stages.

The temperature rises considerably in some very severe cases; generally, however, it is not markedly high.

The bowels are usually constipated, and the bladder becomes contracted.

During the course of the disease there are periods of calm alternating with violent paroxysms.

These latter are easily induced by any sudden disturbance of the patient by loud noises, or by sudden flashes of light into the darkened box. Quietude tends to subdue the paroxysms in a corresponding degree.

During their continuance the breathing becomes more accelerated and difficult, and the surface of the body is bedewed with perspiration; the nostrils are dilated, and the nose protruded.

Course and Prognosis.—The duration of tetanus varies very markedly. Some of the most severe cases have been known to run their entire course in less than forty-eight hours.

In other instances, the animal may live two or three weeks, and then succumb at the end of that period.

As a rule, tetanus runs a more rapid course in thoroughbred horses than in animals of coarser breed, and appears to be of a more active type in excitable horses than in animals less sensitive to external agencies.

In the horse it usually assumes an acute or sub-acute form.

When the disease does not prove fatal, the tetanic condition declines slowly, lasting from three to five weeks or so.*

* In a case quite recently under our notice, the muscles of the near side of the neck of a young pony continued in a state of contraction for

In some very severe cases the breath becomes fœtid before death, and if the mouth be examined, a quantity of slate-coloured epithelium is found on the inner surfaces of the lips, gums, and tongue (Williams).

Morbid Anatomy.—In some instances the spinal cord is found to be congested in varying extent and position, and microscopic examination in some instances reveals pathological lesions, which, though perhaps not characteristic, are nevertheless very marked.

The right side of the heart is sometimes found engorged with blood, and the lungs and other organs sometimes congested.

In some instances, one or more of the muscles may be found ruptured from the violence of the muscular spasm.

Microscopic Appearances of the Nerve-structure.—The perineurium around the nerves proceeding from the seat of injury is sometimes thickened. In a case in which after death a piece of straw was found imbedded in the metatarsal nerve, the latter was found highly congested by Mr. D. Gresswell for some distance from the actual seat of injury.

In the epineurium surrounding the nerve-bundles no special change was noted, but each bundle contained an amount of granular material which stained deeply, and was very probably the result of inflammatory action.

The ultimate nerve-fibrils were altered, and the medullary sheaths of Schwann were shrunken, while the axis cylinders appeared as though split into several segments.

In no instance have we been able to detect any special change in the sympathetic nerves or ganglia.

Sometimes the spinal cord is much altered in certain parts. In one instance, following on very severe injury of

upwards of four months, and after that time gradually relaxed. During the latter three months the animal was perfectly well in every other respect.

the posterior extremity, patches of altered tissue were found in both lateral columns, and in the posterior grey horns of the spinal cord in the lumbar and dorsal regions.

These were less numerous in the cervical region, and stained deeply with hæmatoxylin. They were visible to the naked eye as rounded oblong patches.

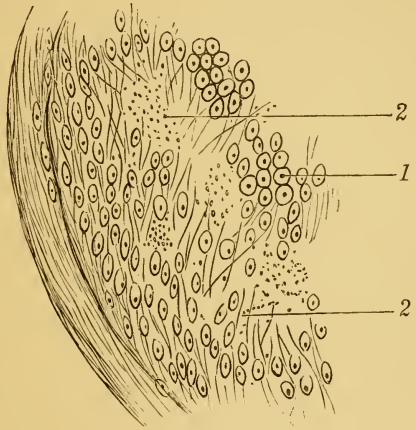


Fig. 5.—Section of peripheral part of the spinal cord of the horse in the lateral column of the lumbar region. 1. White fibres in section. 2. Patches of altered tissue.—*A. Gresswell.*

The cord and its membranes were congested in parts.

Treatment.—The animal should be placed in a large well-bedded and well-ventilated loose box, which should be kept dark.

Strict quietude should in all cases be enjoined, and is of the utmost importance in the treatment of this affection.

Among the many drugs which have been tried for the cure of this disease, no one in particular can be looked upon in the light of a specific.

A moderate dose of some purgative should be administered in the first place, and the bowels afterwards kept open by a laxative diet of mashes and oatmeal gruel.

The wound, if there be any, should be carefully examined, and all irritating matter washed away.

In cases of tetanus following docking, the operation is repeated by some, but is condemned by other authorities. The late Mr. D. Gresswell advocated the repetition of the operation in such cases.

Of the many remedies recommended for the treatment of tetanus may be mentioned—belladonna, hydrocyanic acid, nicotine, aconite, chloroform, chloral hydrate, bromides of sodium and potassium, morphia, Calabar bean, salicine, and alkalies.

Of the Calabar bean and its alkaloid, physostigmina, we cannot speak favourably.

On the whole we have had most success with *nicotine, chloroform, tincture of aconite, caustic potash and morphia.*

Chloral hydrate has also proved of value in many cases.

Belladonna may be given in the form of extract or tincture. If the extract be selected, about one drachm or more may be given three times daily. It may be administered by smearing it over the tongue or by placing it among the back teeth.

Tincture of aconite or morphia are very useful in allaying severe paroxysms, as also is chloroform.

If hydrocyanic acid and bromide of potassium be given, they may be added to the drinking water. Of the hydrocyanic acid, ʒi. may be given three times daily with ʒii.—ʒiv. of potassium bromide.

With regard to the question of slinging, authorities are not of one opinion. We are disposed to advocate slinging in the early stages, in the case of heavy draught horses used for agricultural or other purposes.

In the case of thoroughbred or very nervous excitable horses, the advisability of slinging is questionable. Considering, however, that horses already recovering sometimes fall when the muscles begin to relax, and, struggling violently, bring on the spasmodic contraction more severely, some advise slinging loosely, at any rate, but not so as to irritate or annoy the animal.

Locally, liniment of belladonna and camphor may be applied along the spine. Irritating liniments and mustard are recommended by some, but probably they do as much harm as good. Other authorities recommend the application of ice-bags.

ENZOOTIC PARAPLEGIA — GRASS-STAGGERS.

Definition.—'Is a disease affecting horses feeding on rye-grass at a particular period of its growth, and characterized by disturbance of the innervation, especially by impairment or loss of motor, and more rarely of sensory, power of the posterior extremities' (Robertson).

Nature and Etiology.—Enzootic paraplegia rarely or never occurs when fed on any food save rye-grass at a particular period of its growth; and we believe with Mr. Robertson that it is to be regarded as a different affection from that termed 'stomach or sleepy staggers.' In the disease under consideration, spinal symptoms are the essential features, though cerebral symptoms may be also developed, especially in severe cases.

In stomach staggers, on the contrary, cerebral symptoms are essential features.

This reflex or sympathetic paraplegia does not seem to have any relation to the quantity of the food taken, but only to its quality; and it appears as an enzootic affection where horses are grazed on land where rye-grass abounds.

Cattle and sheep are also liable to become affected through eating rye-grass; and although some seem to regard these morbid states in ruminants as being similar to reflex paraplegia, Mr. Robertson is rather doubtful of any similarity.

According to this authority, 'acute diseases of animals, intimately associated with or resulting from dietetic causes, are all, or nearly all, apparently referable to changes in connection with the cerebral part of the system, and paraplegia alone is not a diagnostic feature of them.'

It must be remembered that ruminants seemingly have a greater power of resistance against most vegetable poisons, and whereas the horse, when grazing, is disposed to eat the flowering stems almost entirely to the exclusion of the foliage of the plants, the ox crops both foliage and seed stems, and the sheep mostly keeps closely to the root foliage. It is, therefore, not to be wondered at if dissimilar symptoms are manifested by these animals.

The immediate cause of the reflex paraplegia seems to be due to the tonic influence of the rye-grass while the seed is being matured.

In some of the features the disease appears to bear a certain resemblance to ergotism.

Symptoms.—The symptoms are gradually developed, and the animal manifests deficient controlling power over the voluntary muscles, especially in those of the hind extremities.

The weakness gradually increases, and the animal reels or staggers.

There is usually no unconsciousness, and in most instances the animal continues in the standing position.

The appetite does not generally suffer, and though the pulse is accelerated and temperature elevated in severe cases, they are nevertheless, in the general way, not altered.

The bowels are generally constipated. Muscular spasms are occasionally developed.

When the animal falls, the muscular spasms are sometimes very severe; consciousness becomes impaired; the breathing becomes stertorous, and death soon closes the scene.

Prognosis and Morbid Anatomy.—The mortality is small, and when the cases are attended to in the early stages death is very rare.

The post-mortem appearances are neither very characteristic nor well made out.

Treatment.—The animal should be removed to a fresh pasture, in the first place.

In most instances recovery then follows without further treatment.

If a purgative medicine be given at first, the animal should be brought up to the stable.

Tonics, such as gentian, ginger, and nux vomica, may follow the administration of the purgative.

CHAPTER XI.

EQUINE ENTOZOA.

THE entozoa which infest the horse are divided into four groups—

I. TREMATODA, or FLUKES.

The trematodes are so called because their bodies are studded with little perforations or suckers.

The word 'fluke' is derived from an old Saxon term signifying anything flat.

Flukes are oval or flat, but they may be spherical, whilst others, such as the Bilharzia, are more lengthened.

This group is divided into several families, according to the number of mouths they possess. The six families recognised are as follow:

1. Monostomidæ.
2. Distomidæ.
3. Amphistomidæ.
4. Tristomidæ.
5. Polystomidæ.
6. Gyrodactylidæ.

The chief flukes recorded as having been found in the horse are—

(A.) **Fasciola Hepatica,** otherwise known by the name of Distoma Hepatica, or the Liver Fluke.

This parasite, although occasionally found in the horse, is more commonly encountered in the ass. Very few cases only are recorded of this worm being found in the horse, and, when present, it generally appears to be harmless.

The life history of this interesting parasite will be more appropriately treated of in works on Bovine and Ovine Medicine.

(B.) Amphistoma Collinsii and Stanleyi.—These species are rarely found in the colon of horses, and are liable to set up intestinal irritation. The fresh specimens are of a brick-red colour, and have been sent from India.

(C.) Gastrodiscus Sonsinonis.—This amphistome, which exceeds half an inch in length and one-third in breadth, was discovered by Dr. Sonsino in the carcases of solipeds which died in the Egyptian plague in 1876.

II. CESTODA, or TAPEWORMS (κεστος, a girdle).

This group includes not only the tapeworms, but also the cystic worms. The members of this group have two distinct phases of existence. In the one, the scolex of the parasite is connected with a so-called cyst or bladder, and is buried in the solid tissue of the host. In the other, the adult tapeworm is found in the alimentary canal.

The adult worm is made up of a number of proglottides fastened on to a scolex, or head, which is fixed on to the mucous membrane of the gut. A fully matured tapeworm is thus in reality composed of a colony of zooids.

Life-history of a Tapeworm.—When the proglottides, or segments of the adult worm, become mature, they pass off with the fæces. Each segment contains a quantity of ripe eggs; these, becoming scattered about, may gain access to the alimentary canal of a suitable intermediary host. In this new situation the six-hooked embryo which is contained in the

egg breaks its shell and obtains its liberty. The immature worm or embryo now passes through the intestinal wall and wanders about until it arrives at a spot suitable for its development into a perfect cyst.

In this condition it will remain and perish, unless its host dies or is killed. In the latter case it may gain access to the intestinal canal of another host fitted for its further existence, and in this case the scolex fixes on to the gut, and the proglottides slowly develop.

It is very rare that the same animal serves as host both for cyst and adult worm.

Some tapeworms have hooks on their proboscides, others are devoid of hooks.

The chief tapeworms of the horse are :

(A.) **Tænia Plicata.**—This worm varies in length from six inches to three feet. It has a short neck, and a body composed of narrow segments, which give to the worm a blunt serrated margin. Its habitat is usually the small intestine, although it has occasionally been found in the stomach. Of all equine cestodes it is the largest.

(B.) **Tænia Perfoliata.**—The length of this parasite is from one to three inches ; it may, however, be five inches long. It may be recognised by the two fleshy lobes, the almost indistinct neck, and the visible narrowing of the segments as they near the tail. The favourite habitat is the cæcum and colon, where this worm is sometimes found in large numbers. Of equine cestodes, it is the most common, and may give rise to serious disturbance.

(C.) **Tænia Mamillana.**—This member of the group is about half an inch long, and has no obvious neck, no lobes, and is made up of wedge-shaped segments with prominent reproductive papillæ. It infests the colon.

Treatment of Tapeworms.—Such drugs as oil of male fern, absinthe oil, or oil of turpentine, should be given.

Cestode Epizooty.—This epizooty occurred among some ponies in South Wales, and among the prominent symptoms were extension of the head, the animals appearing as though suddenly distracted, rubbing the quarters, running and rolling about for a few minutes, then getting up again, and appearing to be well again. If coming down a slope the animals would fall, and in some cases there was lameness in one hind-leg.

Source of the Tapeworms of the Horse.—The cystic forms of tænia placata, tænia mamillana, and tænia perfoliata, are as yet unknown.

Probably their larval forms exist in the bodies of different insects.

Larval Forms of Tæniæ.—Larval forms have received various names, according to their contents, viz. :

1. *A Cysticercus* is a cyst filled with serum, and provided with only one head.

2. *A Cœnurus* is a cyst filled with serum, and provided with many heads.

3. *An Echinococcus* is a primary cyst, which contains, or which gives rise to, secondary cysts filled with serum, each provided with numerous heads.

LARVAL FORMS OF TÆNIÆ OF THE HORSE.

The larval cestodes of the horse are, comparatively speaking, somewhat rare. The one most frequently met with, however, is that of the Tænia Echinococcus.

1. ECHINOCOCCUS VETERINORUM.—This cystic worm is the larval form of the tænia echinococcus, which infests the small intestines of the dog and wolf.

The adult tapeworm is composed of four segments, or joints, and is a little over a quarter of an inch in length. The first joint includes the head, which is about $\frac{1}{100}$ of an

inch wide, and is provided with four suckers, a double coronet of hooklets, between thirty and forty in number, and a central rostellum.

The fourth segment is as long as all the rest of the worm; and is alone supplied with sexual organs.

The cyst, larval form, or hydatid, which has received the name of echinococcus veterinorum, is characterized by almost indefinite increase in size, and by almost indefinite multiplication by the formation of gemmæ.

This cyst, in its early form, is small and globular, with transparent walls and finely granular contents. In its later stages, when it has much increased in size, the walls become thick and the contents fluid.

The colourless fluid and limpid contents contain a quantity of salt, and usually no albumen.

The walls are composed of two coats, an outer, which is fairly thick, transparent, elastic and laminated, and an inner, which is thin, and made out of fine cells, sometimes containing oval or globular refractive bodies.

In some cases the hydatid is only increased in size, but as a rule secondary cysts form in the substance of its walls. The cysts grow—some towards the outer aspect, some in the mid-region, and some towards the inner aspect, and often in connection with the cellular laminæ.

The secondary cysts often repeat in their growth all the characters of the parent hydatid.

This hydatid may be found in various organs and glands of the body, especially the lungs, liver and kidneys, and sometimes in the brain. They vary much in size, sometimes being as small as a pea, and occasionally encountered as large as a good sized cocoa-nut. They may or may not produce symptoms which vary according to the organs affected, and the size and exact position of the cyst.

2. **CŒNURUS CEREBRALIS.**—This hydatid is the larval

form of the tænia cœnurus of the dog. Its full description will be more appropriately treated of in works on ovine pathology.

It is rarely found to affect the horse, one authenticated instance of its presence in the brain only being recorded.

3. **CYSTICERCUS FISTULARIS**, or Long Bladder-worm of the horse, is probably only a variety of the C. tenuicollis of ruminants.

The muscle-flesh of horses is not liable to be infested by cysticerci.

III. NEMATODES, OR ROUND-WORMS.

The nematodes (νεμα, a thread) comprise not only the round-worms, but also the thread-worms and the strongyles (this latter term meaning round or cylindrical).

Included in this group are several families, among which are the

1. Ascaridæ.
2. Cheiracanthidæ.
3. Cucullanidæ.
4. Strongylidæ.
5. Trichinidæ.
6. Oxyuridæ.
7. Trichocephalidæ.
8. Filaridæ.
9. Gordiidæ.
10. Anguillulidæ.

The chief nematodes which infest the horse are the following :

1. **ASCARIS MEGALOCEPHALA**, or Lumbricoid Worm. —This worm is distinguished from the lumbricoid worm of man and pigs by its greater number of caudal papillæ. In length, the male of this species is rarely over seven inches,

but the female may attain a length of sixteen to eighteen inches.

This worm has a smooth body with transverse rings, and may occur in any part of the intestinal canal, although it is especially found in the ilium and small intestine.

With regard to the question how horses become affected, Dr. Cobbold writes: 'It is not likely that any intermediate host is necessary for the growth of the larvæ, prior to their access to the definite host. I have reared the larvæ in impure water and in moist horse-dung up to the size of $\frac{1}{30}$ of an inch. They were then furnished with a completely formed digestive apparatus.' Davaine kept the intra-chorional embryos alive in water for five or six years.

It is noteworthy that the eggs of lumbricoids effectually resist dryness, and it is possible that horses become infected with the ascaris by drinking out of ponds containing sewage matter.

The equine ascaris may prove fatal by setting up enteritis, or by overcrowding it may cause rupture.

Treatment.—Allow nourishing diet, and give internally a brisk cathartic, *e.g.*, aloes, with ʒi. of antim. pot tart., and follow with vegetable tonics, sulphate of iron, and doses of santonine.

Preventive Measures.—See that the water-supply is good, pour boiling water on all worms passed, and attend to the drainage.

2. STRONGYLUS ARMATUS.—This worm has long been known to naturalists, and was formerly described as two varieties, major and minor, which are now proved to be the same worm in different stages of growth.

Description.—The body of the worm is rigid, the head lobular, flattened, truncated, and armed with numerous upright denticles, like a circular saw.

The posterior ray of the hood of the male is three cleft.

The males are about one and a half inches long and the females two inches.

The eggs, which are elliptical and somewhat constricted at the centre, pass from the parent worm and host with the fæces.

In a few days the rhabditiform larvæ are hatched, and in three weeks' time change their skin in mud, and at the same time part with their tails.

They next gain access to the bodies of some intermediary host, where they are still further matured, and from this gain access to the horse.

From the intestinal canal they get into the small vessels of the intestine, and pass on to the larger arterial trunks, producing aneurism, caused by inflammation and thickening of the intima (endo-arteritis).

Lastly, these parasites make their way out of the vessels, and from thence endeavour to regain the colon, where they again change their skins, and, adhering to the mucous lining of the bowel by means of their spines, attain sexual maturity. During their wanderings they are apt to stray.

Verminous Aneurism.—Occurs in a large percentage of horses and asses, and is especially found in the aorta and anterior mesenteric artery.

These aneurisms assume various forms, *e.g.*, thumb-shaped, or bottle-shaped, etc. They may be simple or compound, and vary in size from a pea to a man's head, and occur in animals six months old and upwards. The number of worms in one aneurism varies, and is usually nine to ten, the highest number in one horse being 121.

We append a few extracts of Dr. Cobbold's summary of the results of Bollinger's investigations of this worm:

'The favourite seat of the worm-aneurism is the trunk of the anterior mesenteric artery, directly at its origin from the abdominal aorta. Most frequently that part of the arterial trunk is dilated, from which the

arteria ilia, cæcales, and colica inferior (arteria ilio-cæco colica) arise, less frequently the arteria colica superior at its origin, and the arteries of the cæcum and colon in their course in the meso-cæcum and meso-colon. The verminous aneurism also occurs in the cœliac artery (Bauchschlagader), in the posterior mesenteric artery (gekros-arterie), in the renal artery, and in the abdominal aorta. A horse is not unfrequently afflicted with several aneurisms of this kind at one and the same time. Thus, in one case (described by Bollinger) there were six of these aneurisms affecting the abdominal aorta and its branches in the same horse. The verminous aneurism may occur from the sixth month of life onwards; and with increasing age, the number of horses free from such aneurisms becomes continually smaller.

'The walls of the aneurism are, almost without exception, indurated. In addition to the mesenteric connective-tissue, all the arterial coats, and especially the tunica media, generally take part in this induration.

'The changes in the intima present all stages of progressive and retrogressive metamorphosis, and are the least constant.

'The changes vary from simple induration to ulceration and calcification. In addition to the palisade worms, one almost constantly finds a parietal thrombus contained in the aneurism. It covers the inner walls, either partially or completely, being in the latter case perforated by arterial offshoots. This clot may occlude the artery, and it is not unfrequently continued into the arterial branches (peripherally), or into the aorta (centrally). Amongst the various changes that the clot undergoes, organization of its outermost layer and softening are the most frequent. The constant occurrence of this clot is due to the presence of the worms, to the inflammation, ulceration, and retrogressive affection of the intima, and to the dilatation of the arterial tube.'

3. **STRONGYLUS TETRACANTHUS**, or Four-Spined Strongyle.—This nematode is found in large numbers in the horse, ass, and mule, and though not an uncommon cause of epizooty in this country, is not so frequently met with on the Continent.

These parasites infest the cæcum and colon. Concerning this parasite Cobbold writes: 'The males and females are of about equal size, the largest females reaching nearly three-quarters ".'

In the sexually immature condition the worm occupies the walls of the large intestine, where it gives rise to congestion, ecchymosis, inflammation, and the formation of pus deposits.

The species is readily recognised by its bright red colour,

by the four conical spines surrounding the mouth, by the two neck-bristles, and by the three-lobed long head of the male, and by the posterior three-cleft ray having a rudimentary or fourth branch attached to its outer edge.

The head, when viewed in profile, is truncated, and from above it is round. The body is smooth, and presents indistinct rings.

Development.—The eggs of the worm probably gain access to the intestines of small insects, and the immature form is swallowed by the horse in the water or in the fodder.

The worms then become encapsuled in the mucous membrane of the large intestine, and are about three to six millimetres long when uncoiled.

In this condition the sexual apparatus is not developed; and the skins are cast while the worm is still in its capsule.

Dr. Cobbold, writing concerning the remaining history of the development of these worms, says after escaping the walls of the intestine, the worms re-enter the lumen of the bowel to undergo another change of skin prior to acquiring the adult state. They do this by rolling themselves within fæcal matter of the horse's intestine. In this state they lie coiled up in a kind of cyst or cocoon.

In some districts the worm is not often met with, while in others it is most destructive. Mr. R. Lloyd was the first who recognised this worm as the cause of Welsh epizootic outbreaks.

The worm gives rise to emaciation, colic, diarrhœa, and sometimes to inflammation of the bowels.

Sometimes the intestinal pain is very acute, and the animal rolls and tosses about in great agony. In other instances abdominal pain is not a marked feature.

Treatment.—A purgative should be given in the first instance, and should be followed up by mashes, and afterwards

by good nutritious food and the administration of tonics and stimulants.

Some practitioners recommend the administration of turpentine; but in our hands it has not proved of much benefit.

4. OXYURIS CURVULA, or Pin-worm.—This worm is partly transparent, and is marked with transverse striæ.

In length the males are one and three-quarter inch, and the females from three to four inches, or even more.

This worm has a long subulate tail. The usual habitat is the entire length of the colon, where they are apt to set up local irritation.

The eggs accumulate round the anus; and, according to Cobbold, horses directly become infected with the worm by ingesting the eggs.

Treatment.—Give cathartics and enemas; the latter should be medicated.

5. SPIROPTERA MEGASTOMA, or Large-mouthed Maw-worm.

The length of this species is from one-third to half an inch, the females being a little longer than the males.

Dr. Cobbold thus describes this worm: 'The males are one-third of an inch and the female half an inch long. Around the mouth are four thick horny lips, the dorso-ventral being the larger. The tail of the male is spirally twisted, and supplied with lateral bands, supported by three or four ribs. It carries two curved spicules of unequal size. There are five pairs of caudal papillæ, and the tail is bluntly pointed in both sexes. The vulva of the female is about one-seventh″ below the head. The eggs are linear, narrow, and possess thick shells.'

The parasites are found in tumours in the walls of the stomach, especially at the cardiac end; the tumours usually contain a bundle of worms together with pus.

The tumours are apt to rupture, and the contents escape into the peritoneal cavity.

6. **SPIROPTERA MICROSTOMA.**—This species, which may probably be regarded as a variety of spiroptera megastoma, is found free in the stomach, and does not appear to be productive of injury.

7. **STRONGYLUS MICRURUS,** or Lung-worm.—This parasite, although so productive of bronchitis in calves, rarely occasions serious results in solipeds. However, it is not unfrequently found in the bronchial tubes of asses and horses on post-mortem examination.

8. **STRONGYLUS GIGAS,** or Renal Strongyle.—This worm has been found in the kidney of the horse. In 1792 it was observed by M. Chabert, and reported or witnessed by Rudolphi and Leblanc.

9. **FILARIA LACRYMALIS,** or Small Eye-worm.—This thread-worm has been found between the lids and eyeball of the horse.

The males are half an inch long, and the females two-thirds of an inch. It is viviparous.

10. **FILARIA PAPILLOSA,** termed by some Filaria Oculi.

The males are two to three inches long, the females five to seven inches.

Description.—The head is broad; the mouth wide, and surrounded by a ring of chitine, and possesses two denticles. On the neck are two papillæ, near the middle line; besides sixteen caudal papillæ, eight on either side. The tail of the male is spirally twisted; the tail of the female slightly curved.

This worm infests the globe of the eye and its tunics, and is often contained in a cyst within the cornea. It is also found in the thorax, abdomen, membranes of the brain, muscle, and cellular tissues.

Dr. Manson considers that the eye is not the usual habitat for this parasite.

The tracks of this worm may be readily seen.

11. FILARIA SANGUINIS EQUI.—These microscopic nematodes were found by Dr. Sonsino in the blood of an Egyptian horse.

IV. ŒSTRIDÆ, OR GADFLIES.

GASTROPHILUS EQUI, or Common Gad-fly, or Œstrus Equi.—**Description.**—The bot, which is the larval form of the gad-fly, has eleven segments arranged in double rows, and is of a red colour. The head is narrow and pointed, and the mouth is small, and placed between two large hooks or maxillæ. Between the hooks are two horny plates, called mandibles. The tail is broad, truncated, and flattened at the end.

Development.—The gad-fly attacks the animals in late summer to lay its eggs, and this is done by fixing them to the neck, shoulders, and inner part of the fore-leg, especially about the knees, by means of a sticky or glutinous secretion.

The horse licks these parts with his tongue, and in less than three weeks after being first deposited the larvæ escape.

From the tongue they gain access to the stomach, where they fix on to the mucous membrane by means of their hooks.

When they attain their full size they become loose, and escape with the fæces and bury themselves in the soil, where they are transformed into pupæ.

Having remained in the earth about six or seven weeks, they emerge from their pupa cocoons perfect dipterous insects.

The *male* insects die, and the females live long enough to deposit their eggs, which are generally about forty in number.

The *bots* of the horse live in the stomach in winter months, leave in spring or early summer, and remain in the soil six or seven weeks.

The *fly* is developed in June to September, and after the latest females have appeared all perish in October.

The bots are as a rule comparatively harmless, but cause injury where great numbers are aggregated together. They may cause rupture, perforation, or tetanus.

Treatment.—Weak carbolic solutions, cathartic medicine, and ethereal solutions may be given.

GASTROPHILUS HÆMORRHOIDALIS. — The larvæ are found attached to the walls of the rectum.

GASTROPHILUS NASALIS. — The larvæ attach themselves to the duodenum, near the pylorus.

CHAPTER XII.

TOXICOLOGY.

A KNOWLEDGE of the actions of, and symptoms produced by, certain drugs and reagents, recognised in toxicology under the term 'poisons,' is of great importance to the veterinary practitioner.

For a full account of the manner in which different poisons may be absorbed into the system, and for the detailed methods by which they may be detected, we must refer the reader to such works as 'Taylor on Poisons,' or Dr. Tanner's little work.

Here we intend to treat of arsenical poisoning, acute and chronic; lead poisoning, acute and chronic; and also, as shortly as possible, of poisoning by antimony, corrosive sublimate, hellebore, yew-tree foliage, opium, savin, bryony, water drop-wort, euphorbium or spurge, and cantharides.

ARSENICAL POISONING.

This metallic poison is usually administered to the horse in the form of arsenious trioxide, or common white arsenic.

This substance is very commonly given by labourers and waggoners, and more rarely by grooms, in certain parts of

the country. When given in excessive doses it is generally through ignorance, but in rare instances may be administered with criminal intent.

The drug is usually given in bolus, made up with soap, tar, or sulphur, or indeed with any suitable substance. Sometimes it is administered as a powder in the food or water.

Symptoms and Post-Mortem Appearances.—The following accounts will serve to indicate some of the more important symptoms and post-mortem appearances of arsenical poisoning in the horse. We propose to draw attention, firstly, to an outbreak which occurred on a Thursday morning, some time ago, on a large farm on the Wolds, near Louth.

When called in, at 3 a.m., four cart-horses were found to be in a very dangerous state. They were fine heavy animals, in excellent condition, and on the previous day had shown no signs whatever of ill-health. Their restlessness first attracted attention about 12 or 1 a.m. When examined, they were observed to be breathing rapidly, the respirations varying from 30 to 40 per minute. The arteries were in a state of extreme contraction; the submaxillary artery, indeed, feeling like a mere thread. The rate, also, was much accelerated; in one case, the pulse reached 108; in another, 96; in a third, 80 per minute; while in a fourth it was entirely imperceptible. The weakness and irregularity of the pulse were very marked. The temperature varied: the highest was 105·5°; and in the other three it reached 104°, 102·5°, and 101·8° respectively. All four animals were purging violently, with extreme tenesmus, and they were in great pain. They got up and down alternately, rolled over and over, and manifested other signs of intense agony. Their extremities were cold. and the visible mucous membranes injected. The eyes

were staring, and the pupils much dilated. There was entire loss of appetite and extreme prostration. Eructations of gas frequently passed from the stomach and intestines.

The horse with the imperceptible pulse died at 10 a.m., a second died at 4 p.m., and a third at 10.30 p.m. Before death the animals became still more restless, the pulse was weaker, and finally imperceptible; the mouth became clammy, and the breath fœtid, and they succumbed at length in a state of extreme agony and collapse. One became quite comatose before death, and another had tetanic spasms; the remaining animal recovered. For a time this horse remained so weak and debilitated as to be incapable of rising without assistance; at length, however, he made a gradual and apparently complete recovery, but was not able to resume work for three or four months.

On the following day a careful post-mortem examination was made in each of the three fatal cases. *Thorax*: The lungs were found to be much congested; the endocardium of the left ventricle was highly inflamed, and patches of ecchymosis were found studding its surface in several places; the left ventricle was full of black coagulated blood. *Abdominal cavity*: The stomach was found to contain much undigested food, and the contents were tinged with blood. The cuticular portion had several small patches of redness, and its walls were eroded, in one of the three cases, near its junction with the villous portion. The villous portion was of a dark-brownish-red hue, being intensely inflamed, and covered in parts with a film of coagulated lymph.

It was blackened in patches, and in parts was of a dirty yellow colour; while in some places the walls were much eroded, forming many large eschars, and in other places the mucous membrane was raised in the form of small

bullæ, these being produced by effusion of blood beneath the lining membrane.

In one of the cases there were two almost complete perforations through the walls.

The small intestines contained much dirty grey mucous, and presented patches of acute inflammation. The large intestines also showed signs of inflammation, the mucous membrane being reddened in patches. The peritoneum showed spots of ecchymosis in various places.

The brain was not examined.

Though the waggoner denied having administered arsenic, it was afterwards elicited that he had given to each of the horses a quantity of arsenious acid, made into balls by mixing it with tar.

It was calculated that each horse received about ʒiv. to ʒi., but it was not possible to ascertain the exact amount.

This he had given at about 8 or 9 p.m. on the previous day, April 11th.

The symptoms here recorded, and the pathological lesions found, are characteristic of acute arsenical poisoning; and arsenic in abundance was found in the contents of the stomachs. But the effects of arsenic vary much with the amount given, and with the method of administration.

About 11 p.m., on June 20th last year, we were summoned to a team of four cart-horses belonging to a farmer who lived a few miles from Louth. The symptoms in these cases were much less severe than those above described.

The pulses were 90, 72, 60, and 62 respectively, weak and fluttering. The submaxillary artery was contracted, and felt like a mere thread. The respirations were also increased, the legs and ears cold, and there was total anorexia.

One of the animals, an old cart-mare, became violently purged about 12 p.m., and the liquid fæces were blood-

stained; the mare died at 2 a.m. on the following day, and the autopsy revealed pathological conditions somewhat similar to, but much less marked than, those described in the previous case.

The other three animals made a gradual recovery, and were soon again at work.

It was ascertained in these three instances that arsenic had been administered in the form of powdered arsenious trioxide. The waggoner had for some time previously given to each of his horses every night as much as he could place on the end of a large pocket-knife. But on the night when the horses were so suddenly affected he had given an extra dose to each, three or four hours previous to the appearance of symptoms of poisoning. Probably each horse had received from two to three drachms.

Several months afterwards we took the opportunity of examining two of the horses again; both had an intermitting pulse, irregular in rhythm and volume. The foreman told us that they had never regained their previous strength. Their appetite was somewhat impaired, and when out at work they were far more easily fatigued than the other horses, and breathed violently when any additional strain was put upon them.

After the administration of vegetable and mineral tonics, they made considerable improvement. Their condition was doubtless in part or wholly due to *the effects of the arsenious acid on the endocardium lining the left ventricle of the heart, and, indeed, a distinct mitral murmur could be heard in one of the cases. This murmur was systolic in time.* That arsenic, when given in solution, acts much more rapidly and powerfully is shown by the following record of nine cases of poisoning which occurred in Mr. D. Gresswell's practice some years ago.

One of the waggoners on a large farm, having obtained a

pound and a half of powdered arsenious trioxide, stirred it up in a tub of boiled linseed gruel. This was served out equally to nine horses on their return from work at two o'clock in the afternoon. Very shortly afterwards, viz., about half an hour, the horses began to manifest considerable uneasiness, and the owner in consequence telegraphed to Louth. When Mr. Gresswell arrived he found that eight of the horses had died; but the ninth case, which had an almost imperceptible pulse, recovered under careful treatment.

The time that had elapsed between the administration of the poison and the death of the animals varied from one hour and fifty-five minutes to two hours and thirty minutes.

The horse which recovered remained very debilitated for many weeks afterwards.

Each horse, it is believed, received from an ounce and a half to two ounces of arsenious trioxide, partly in a state of solution, partly in a state of mechanical suspension.

We might record many other cases, but the above will suffice to illustrate the main symptoms produced in the horse by large doses of arsenic.

We may now say a few words concerning chronic arsenical poisoning in the horse.

In February last we were called to see a valuable seven-year-old hunter, belonging to a gentleman residing a short distance from Louth. The horse had an excellent appetite, but was in poor condition. On examination the pulse was found to be fairly strong, but irregular, losing one beat in every six. The respirations were somewhat accelerated. We were informed that when galloped, even for a short distance, the horse breathed laboriously, and could only with difficulty be induced to go beyond a fair trot.

It was ascertained that for many months previously the late groom had given to the horse small doses of arsenic

at regular intervals. The symptoms presented were to be attributed to this practice, as the horse always enjoyed perfect health previously, and made much improvement when the groom left.

Diagnosis.—The history of the case will aid us in forming a diagnosis in some instances.

The thread-like character of the pulse, and the purging with extreme tenesmus in severe cases, will assist us in diagnosing acute arsenic poisoning from enteritis.

Detection of Poison.—In the solid state arsenious acid may be detected by the following tests:

(1.) When heated on platinum foil arsenious acid produces a white vapour, and is entirely volatilized.

(2.) If some of the powder be heated in a small test-tube it will be sublimed, and small octahedral crystals, visible to the naked eye or by a lens, settle on the sides of the tube.

(3.) If arsenious acid be mixed with freshly burnt powdered charcoal, and heated in a small test-tube, a ring of shining metallic arsenic of a grey colour will be found on the cool portion of the tube, and an odour of garlic is perceptible.

If this deposit be heated it will become oxidized, and octahedral crystals of arsenious acid formed.

Further, the arsenious acid may be dissolved in distilled water and the liquid tests applied to the solution.

In solution arsenious acid may be detected by several tests. The solution of it in water is colourless, almost tasteless, and has a very slightly acid reaction. If a few drops be evaporated on a glass slide and examined by the microscope, numerous minute, and mostly imperfect, octahedral crystals of an amorphous deposit will be seen, presenting triangular surfaces by reflected light.

(1.) The ammonio-nitrate of silver (prepared by adding a few drops of liquor ammoniæ to a solution of nitrate of

silver till the brown oxide of silver at first precipitated is nearly redissolved) throws down with arsenious acid a rich yellow deposit of arsenite of silver.

(2.) The ammonio-sulphate of copper (formed by adding liquor ammoniæ to a solution of sulphate of copper till the bluish-white oxide of copper is almost redissolved) produces a pale-green precipitate (arsenite of copper).

Care must be taken not to add too much of the test in the first instance, otherwise its blue may overpower the green of the precipitate.

(3.) Sulphuretted hydrogen water throws down a yellow deposit of sulphide of arsenic.

It is better, however, to use pure and well-washed sulphuretted hydrogen, generated in the usual manner.

In performing this test care must be taken that the liquid to be examined is not alkaline, or no precipitate will be produced, even though arsenic be present.

For this reason yellow sulphide of ammonium will not precipitate arsenic until acidified with a mineral acid.

There are several other processes for the detection of arsenic which are of great value, especially when mixed with foreign substances, viz.: (1) Marsh's process; (2) Reinsch's process; (3) the method for detecting the poison in an organic mixture.

Marsh's Test.—This test is founded on the fact that arsenious acid is decomposed by nascent hydrogen, forming arseniuretted hydrogen gas.

This vapour has the following properties:

It burns with a bluish-white flame, and gives off a white smoke (arsenious acid) which has an odour of garlic.

If a piece of glass or porcelain be held in the flame, a blackish metallic stain, consisting of metallic arsenic, will be deposited upon it. This stain might be confounded with one produced by antimony under similar circumstances.

Antimoniuretted hydrogen, however, does not burn with the odour of arseniuretted hydrogen.

The antimonial deposit is sooty, and has not a metallic lustre, and is darker than that of arsenic, which is of a browner hue. The arsenical deposit is, moreover, readily dissolved by a solution of chloride of lime (bleaching powder), whilst that of antimony is not affected.

To the stain may be added a few drops of nitric acid, which will be found to dissolve the arsenic.

If this be now allowed to evaporate, and the acid be neutralized, a few drops of nitrate of silver will give a brick-red precipitate of arsenate of silver.

Marsh's test may be carried out in the following way :

A flask is furnished with a cork, through which pass a funnel and a long test-tube drawn to a point, and the funnel is made to reach almost to the bottom.

Several pieces of pure metallic zinc are now introduced, and then some dilute sulphuric acid is passed through the funnel.

In this way hydrogen is produced, and escapes by the test-tube. When all the air has been expelled the gas should be ignited as it escapes, and a piece of cool porcelain held over the flame.

If there be no deposit, it is plain that neither the zinc nor the sulphuric acid contains arsenic.

A portion of the suspected fluid is next introduced into the flask by the funnel, and the escaping gas again tested.

Should a stain, giving the characters alluded to above, be produced, the fluid contains arsenic in some form or other.

Reinsch's Test.—The liquid suspected is boiled with one-sixth to one-eighth of its bulk of pure hydrochloric acid, and a bright slip of metallic copper is introduced.

If arsenic be present, the copper will be coated with it in the form of an iron-grey deposit.

The copper should be removed, washed with distilled water, and dried between the folds of blotting-paper cut into slips; it should then be placed in a dry tube and heated.

If arsenic be present, arsenious acid will be sublimed and deposited on the sides of the tube in the form of minute octahedral crystals.

These may be dissolved in water and tested in the usual way. Before applying this test, the hydrochloric acid, as well as the copper foil, must be examined, to make sure of their purity.

This is easily effected by boiling the copper with a mixture of the acid and distilled water before adding the suspected liquid.

Method of Detection of Arsenic in an Organic Mixture.—We think the most satisfactory method of detecting arsenic in an organic mixture is to boil up the suspected matter with a large quantity of pure strong hydrochloric acid. The mixture is afterwards filtered, and the arsenic is precipitated by passing a current of sulphuretted hydrogen through the filtrate. The arsenic is then thrown down as sulphide, and it may be estimated in the usual manner.

Treatment.—The chemical antidotes which may be given in acute arsenical poisoning are hydrated sesquioxide of iron, or hydrate of magnesia.

In acute cases, hot rugs should be applied to the abdomen.

Stimulants may be freely given every two or three hours.

Mucilaginous drinks of linseed and oatmeal gruel, and albuminous fluids, may be given with port wine or brandy.

In the treatment of chronic arsenical poisoning great care is necessary. Vegetable and mineral tonics, especially iron, with good nutritious diet, should be administered internally.

LEAD POISONING—PLUMBISM—SATURNINE EPILEPSY—LEAD PALSY.

Lead poisoning is generally confined to certain districts where lead-smelting is carried on, but it may also occur in horses from ingestion of lead paint and splinters of bullets which are scattered about near rifle-targets.

The lead contained in paint, when ingested, is acted upon by gastric juice, and being thus converted into soluble salts, is absorbed into the system.

In its pure metallic form, lead appears to be devoid of toxic properties; and it is well known that in the form of shot it is used by dealers of low principles to alleviate the symptoms of broken wind in horses they wish to sell. Some of the metal, however, is very probably converted into soluble salt, and being absorbed, becomes incorporated with the tissues.

In some instances lead poisoning has been due to boiling food in vessels used for containing lead preparations.

It is probable also that lead may be absorbed by water conducted through leaden pipes, and this is more especially likely to be the case, when the water is highly oxygenated, or contains organic matter.

Polluted shallow well-waters are more dangerous in this respect, because they act on lead violently and continuously, and therefore leaden pipes should never be used.

Lead when absorbed into the system leaves very slowly, and may be retained many weeks after the animal has ceased to ingest it in its water or food.

Lead poisoning may be acute, when it is sometimes spoken of as 'saturnine epilepsy,' in which coma, delirium, or convulsions are manifested, or it may be chronic.

ACUTE LEAD POISONING.—After ingestion of a large amount of lead, the appetite becomes impaired; there is

copious discharge of saliva; the mucous membrane of the conjunctiva is injected; the animal is dejected, and sometimes the tongue is protruded.

The limbs are often drawn under the body; the head is depressed; the back is arched, and the coat may be rough and staring.

In some instances the animal speedily becomes comatose, while in other cases he is very violent, dashing his head about wildly.

Spasmodic contractions and twitchings of the muscles are often developed.

The breathing is quickened and difficult, and the temperature is usually somewhat elevated. The pulse is sometimes accelerated, and the artery hard and the volume diminished, while in other instances the frequency is not increased and the volume is fuller.

The bowels are usually constipated, but there may be diarrhœa and fœtid stools.

Pain is often, but by no means always, manifested.

In Wales, roaring is said to be the first symptom of lead poisoning in the horse.

CHRONIC LEAD POISONING. — The symptoms of chronic lead poisoning are produced by the ingestion of lead in small quantities over an extended period of time.

This form of poisoning is chiefly met with in the neighbourhood of smelting works, where particles of lead or its salts are disseminated over the grazing pastures, and also probably where lead enters the system through contaminated water-supply.

We are not aware whether any cases proceeding from the latter cause have been described, but some time ago a suspicious case came before our notice.

The appetite is impaired, and the coat becomes unhealthy,

rough, and staring. The bowels are irregular; sometimes constipated, sometimes loose.

In most instances a peculiar blue line is observed on the gums where the teeth are implanted; but in the parts between the teeth this line is not present. This symptom is mostly regarded as diagnostic, but sometimes it appears to be very faint or absent.

Roaring is a symptom generally present, and is probably to be attributed to a paralytic condition of the nerves supplying certain muscles of the larynx (*vide* 'Roaring'). The muscles often twitch and contract spasmodically.

Difficulty of locomotion and paralysis, especially of the hind-limbs, are also liable to become developed in chronic poisoning by lead.

Sometimes the joints become swollen and the flexor muscles of the fore-limbs contracted, probably from paralysis of the extensor muscles of the limbs.

The animal thus appears to stand on its toes, and when it walks, it seems to knuckle over on the fetlocks.

The paralysed muscles may undergo atrophic and degenerative changes.

Diagnosis.—Acute lead poisoning may be mistaken for gastric and enteric disorders, accompanied with paralytic symptoms.

In lead poisoning, however, the muscular twitchings and fits are more frequent and more distinctive. The tucked up condition of the abdomen, the discharge of saliva, and the grinding of the teeth are also features indicative of poisoning by this substance.

In the chronic form of poisoning, which may be mistaken for articular rheumatism of a severe type, the paralytic features, the irregular recurrence of muscular twitchings and spasmodic contractions, as well as the 'blue line' when formed, are sufficiently indicative.

In articular rheumatism, the character of the local swellings and the tendency to metastasis, as well as the acute pain and its persistence, are diagnostic symptoms.

Morbid Anatomy.—*In Acute Cases,* the muscles are found both in a softened and blanched condition. The lungs and trachea are often inflamed. The mucous membrane of the alimentary tract shows alteration of structure, and is sometimes softened and pulpy over a considerable area, owing to infiltration into sub-mucous layer.

Sometimes in parts there are blood extravasations of variable size and extent. The muscular tissue of the heart is softened and flaccid.

Ecchymosis under the endocardium is not present, whereas in acute arsenical poisoning, it is, we believe, *almost invariably found after death.*

The membranes of the brain are often congested.

In Chronic Cases the mucous membranes of the intestines and stomach present patches of ecchymosis and the cæcum and colon often show gangrenous patches.

The swellings in connection with the joints are composed of a collection of clear fluid outside the joint cavity.

Method of Detection of Lead.—The presence of lead in solution may be demonstrated by :

(1.) On passing sulphuretted hydrogen through it, or on adding a few drops of sulphide of ammonium, a black precipitate is formed.

(2.) A white precipitate results from the addition of liquor potassæ or liquor ammoniæ.

(3.) Dilute sulphuric acid gives a similar precipitate, which is insoluble in nitric acid.

(4.) Iodide of potassium gives a bright-yellow deposit (iodide of lead).

Method of Detection of Lead in the Tissues or the Urine.—The tissues should be dried and incinerated in a porcelain vessel.

The ash should be heated with a small quantity of strong nitric acid, and then evaporated to dryness. The nitrate of lead thus formed may be dissolved out of the residue by water, and filtered.

A portion of this liquid, evaporated on a slide, will yield crystals of nitrate of lead, which may be identified by :

(1.) Covering them with a solution of iodide of potassium, when they acquire a brilliant yellow colour.

(2.) Turning black on the addition of ammonium sulphide.

The remainder of the liquid, after filtration, may be treated with a current of sulphuretted hydrogen gas.

A brown colour or a brown precipitate, not readily dissolved by nitric acid, indicates the presence of lead.

The metal itself may be obtained, if necessary, by plunging zinc into a portion of the acidulated liquid (Taylor on Poisons).

Absorption of Lead.—Concerning the absorption of lead, in acute and chronic poisoning by this substance, Dr. Taylor says both in acute and chronic cases the metal, in some form, is found more or less in all the soft organs of the body.

The blue line on the gums affords an instance of its deposition in these parts, the colour being probably due to the conversion of the deposited lead into sulphide.

Lead was found by Tiedemann in the blood of poisoned animals ; and Professor Cozzi found it in the blood of a person labouring under lead colic.

The urine appears to be the great channel of elimination ; and Taylor found traces of it in the milk of a cow poisoned by lead paint.

Dr. Letheby, in a case of chronic poisoning, detected lead in the brain, muscles, and intestines, as well as in the blood and serum found effused into the ventricles of the brain; but none was discovered in the bile or urine.

Dr. Inman detected it in one case in the cerebellum.

M. L. Orfila states that he found it in the tissues as long as eight months after the withdrawal of the poison; and the facts connected with the slow disappearance of the blue line from the gums, on poisoning with this metal, render it probable that it may be detected after the lapse of one or two years.

Treatment.—In cases where the symptoms are rapidly developed from the ingestion of lead from paint or bullet-spray, it is best to administer such agents as enter into combination with lead, forming insoluble salts.

With this view, sulphate of magnesia, or soda with sulphuric acid and sulphur, may be given.

Lead sulphate, which is the most insoluble of all the salts of lead, and lead sulphide are thus formed. Being insoluble, these salts are therefore not absorbed.

If there be much pain, opium and hyoscyamus may be given also.

Should the pain continue, woollen cloths wrung out from hot water should be applied to the abdomen, and hypodermic injections of morphia may be administered.

The sulphuric acid should be repeated every three or four hours for two or three days, in order to prevent the solution of the remaining lead by the gastric juice.

The bowels should be kept in a lax condition by the administration of saline purgatives for a few days, and diluents may be freely given. In addition, the kidneys may be acted upon by the addition of nitric ether, or other mild diuretics.

After a day or two, tonics, such as gentian, quinine and nux vomica, may be regularly given.

In the chronic forms of lead poisoning, where this agent has accumulated in the system, a moderate dose of some purgative should be given in the first instance, and this should be followed up by the administration of potassium iodide.

The iodide, after its absorption in the blood, combines with the lead and forms iodide of lead, which, re-entering the circulation, is eliminated with the urine.

The dose of iodide should be small at first, as when it combines with the lead the phenomena of acute lead poisoning may come on, from the large amount of iodide of lead formed and passed into the circulation.

In some cases of valuable horses, if paralytic symptoms remain, galvanism may be tried, as in man.

In all cases of lead poisoning it is of first importance that the animals be moved from the source whence this agent is derived.

ANTIMONY POISONING.

Antimony is occasionally administered to the horse in the form of tartar emetic, or as chloride or butter of antimony. These salts are not nearly so frequently given as they once were, and the practice, we believe, is now considerably on the decrease.

The symptoms are those of an irritant rather than of a corrosive poison.

There are frequent attempts at vomition, and in some cases this is complete.

In severe cases the pulse becomes weak and fluttering, and almost or quite imperceptible.

There is great prostration and gradual loss of consciousness in severe cases, followed by death.

YEW-TREE FOLIAGE.

Many instances of death from browsing on the leaves of the yew-tree (Taxus Baccata) have been recorded in the horse.

After death, which in some cases took place from two to three hours after the ingestion of the foliage, the stomachs were found contracted and inflamed.

Method of Detection.—Fragments of the leaves or the berries may be found in the stomach after death.

The apex of the leaf of the yew is not so pointed as that of the savin; and the yew-leaf does not possess the peculiar odour of savin when rubbed.

Yew-berries are of a bright red colour, and green at the top, allowing a hard brown kernel to be seen.

The juice, which reddens blue litmus paper, has a nauseous sweetish taste.

Treatment.—The method of treatment to be adopted is the internal administration of oleaginous purgatives and diffusive stimulants, such as the aromatic spirit of ammonia and nitric ether in linseed gruel or water.

CORROSIVE SUBLIMATE

Is sometimes given in injurious doses, causing loss of appetite, salivation, pawing, looking at the flanks, rolling, profuse perspiration, rapid, weak, thready pulse, violent purging, tenesmus, convulsions, and death.

After death the stomach is much inflamed, and in patches intensely so.

The whole intestine is likewise inflamed, and gangrenous in patches.

Antidotes, as white of egg mixed with water, or thick starch or arrowroot, may be given.

HELLEBORE.

Black hellebore, or the Christmas rose, and white hellebore, or veratrum album, are both poisonous when administered in large doses.

The action of the latter is similar to that of the former, but is more irritant.

Hellebore is occasionally administered by grooms to horses under their charge, as they suppose it to have a valuable alterative effect.

Symptoms.—In a case recently under treatment by us, two drachms of the pulverised root of the white hellebore were given by the groom to a carriage horse.

When called in, the head of the animal was protruded; the pulse was much accelerated, and varied from 90 to 100 beats per minute; the respirations were considerably quickened; the extremities were deathly cold, and there was marked nausea, and frequent attempts at vomition. The appetite was completely lost for forty-eight hours, after which it gradually returned, and the animal made a slow, but complete recovery.

The late Mr. D. Gresswell, who had a large number of cases of poisoning by hellebore under treatment at different times, observed complete vomition in some instances, and was of opinion that if given in large doses this drug generally causes actual sickness.

Method of Detection of Hellebore.—Powdered white hellebore-root has a reddish-brown colour resembling jalap.

Nitric acid turns it a red colour, rapidly passing to a dark brown.

Iodine water gives it a bluish-grey tint, which is only slowly brought out. Sulphuric acid turns the powder to a brown-black.

Diagnosis.—Hellebore poisoning is very frequently mistaken for choking by the uninitiated; but the history of the case, when that is to be obtained, and the character of pulse, will at once clear up any doubts.

Treatment.—A good moderate dose of some aperient medicine should be given in the first place, and this should be quickly followed up by the administration of stimulants and tonics.

The diet should be moist and nutritious.

After recovering from the effects of the poison, tonics should be administered for a week or two.

OPIUM.

Opium poisoning is of rare occurrence in the horse.

This drug, however, is sometimes administered by dealers and others in order to prevent kicking and restiveness in horses they wish to sell.

A few weeks ago we were called in to see a black mare, aged eight years, to which the owner had administered one ounce and a half of Turkey opium.

When called in, on the day following the administration of the drug, the mare was found to be in a very dull, dejected condition, and the pulse was very feeble and soft, and the artery very compressible. The number of beats was 84 per minute, and the temperature reached 102·5°.

The pupils were contracted, and there was total loss of appetite, the mare refusing all food and drink. The breathing was slow, but not stertorous, and the nasal mucous membrane was of a dark brownish hue.

The symptoms remained unabated for three days, during which time the animal continually moved round and round in the box. On the fourth day the pulse began to regain vigour; but recovery was not complete before the lapse of a week.

SAVIN

Is sometimes given by grooms and others, with the idea of improving the condition, and has often caused death.

It is said that the presence of savin can best be detected in the stomach of the horse by the black currant-leaf-like smell of the contents when boiled in a little water or beaten up in a mortar.

When seen under the microscope, the leaves have sharply acuminated apices.

BRYONY.

Bryony (*Bryonica dioica*) is often given by horse-breakers to young horses, with the idea of improving the condition.

Although this drug excites the animal, and for a time is said to improve its condition, it is nevertheless decidedly poisonous; and when the transient effects are over, depression of energy and corresponding loss of condition follow.

WATER DROP-WORT.

The water drop-wort (*Œnanthe crocata*) is a plant commonly growing in ditches and marshy localities. This

plant is not often eaten by horses; but brood mares, with appetites vitiated by their being in foal, have been poisoned by eating it.

As antidotes, vinegar and gruel are recommended.

EUPHORBIUM.

Euphorbium, or spurge, is one of the ingredients in the farrier's blisters, and has caused many deaths from the great irritation which it has set up.

CANTHARIDES.

Cantharides, or the Spanish fly, is sometimes administered as an excitant to the horse and mare at the breeding season, and, owing to the large amount given, has been the cause of many deaths.

It is said that a drachm, and, indeed, sometimes a much less amount, will kill almost any horse.

The drug, which is a purely irritant poison, is administered in the pulverized condition in the form of a ball, or as the tincture.

When given as a ball, some portions of the fly may often be detected as little glittering particles which are apparent on the sides of the fæces in hot water.

In cases where cantharides has been inadvertently or wilfully administered in too large doses, oily and demulcent liquids may be given in large quantities.

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